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HISTORY OF CONCEPTIONS OF ETIOLOGY AND THERAPY OF DIABETES MELLITUS, PAST AND PRESENT *

A. A. HEROLD, M. D.
SHREVEPORT

It may seem to many of you that such a title will be dull and useless, but to me and others interested in this subject, past ideas of causation and treatment of this disease are fraught with much of interest —interesting because of changes in theories and also of indications for treatment and how we may avoid those dreaded complications which arise through improper or insufficient treatment or neglect.

As Chairman of this section, I feel that much is in order to be said, much prophesying may be done and, in this review, we find where ideas change and often revert to former thoughts.

In a paper read by me before the American College of Physicians in 1928, I traced the ideas of etiology from the classical experiments of Claude Bernard, during which he demonstrated the famous "piqûre" or puncture of the tip of the calamus scriptorius in the fourth ventricle, causing glycosuria, with depletion of liver-stored glycogen, which may be designated as the starting point of thinkers, searching for rational diabetic therapy. I referred, also, to the work of Conrad Brunner, in 1682, when he attempted total pancreatectomy on dogs, and to the successful experiments of Von Mering and Minkowski, two hundred and seven years later. And I mentioned, also, the oral substitutes at that time and which I shall refer to later in this paper.

THERAPY

A paper of this scope would not be complete without reference to past therapy and a summary of the ideas of the best clinicians as to the merits or demerits of the various preparations advocated in the treatment of this disease, which has been recognized as a clinical entity since the days of the famous Egyptian Papyrus Ebers of approximately 1,500 B.C.

It is interesting to note the many therapeutic measures and drugs which have been recommended and used to reduce hyperglycemia or to clear up, in part at least, glycosuria. For centuries, in some parts of Hungary, the peasants have known that a decoction or tea of certain blueberry leaves helps the polyuric. On this information, Dr. F. M. Allen, in cooperation with his chemist, has experimented with the preparation, which he has named myrtillin and which was placed on the market. While no extravagant claims have been made for it, it has been found to be of some value in preventing the rise of a normal blood sugar. Along similar lines, Dr. E. A. Bertucci has reported definite lowering of hyperglycemia with a preparation made from the thistle.

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For many years, there has been a tablet of yeast on the American market and much has been claimed for it. I recall an associate, with whom I officed in the early days of my practice, himself a diabetic, who claimed to have noted beneficial effects from it, both personally and in his practice. A few years ago, German manufacturers flooded the market with many such preparations, but they do not seem to have made an impression. However, Joslin, in his book on treatment, mentions that "a substance similar to insulin has been prepared from yeast by Winter and Smith and from various vegetable sources by Collip." Prof. von Noorden, in his lectures delivered in this country, in 1905, stated:

"About fifteen years ago Prof. Binz and Dr. Graser discovered that the fruit of the East Indian plant, *Syzygium jambulanum*, possesses the property of strongly reducing phloridzin diabetes. . . . I have, myself, employed and studied the effect of jambul preparations on cases of severe and slight diabetes; the results showed that, in some cases, a marked effect on the glycosuria was, without doubt, actually obtained."

It may be mentioned as of historical interest that von Noorden mentions as the drugs most potent in diabetes, as considered in 1905, opium and its alkaloids, salicylic acid and similar substances (including aspirin) and antipyrin. Joslin, also, in an early work, while deprecating the too ready use of drugs by the profession, mentions opium and aspirin and concludes:

"Organic preparations made from the pancreas and the pancreatic gland, itself, have been frequently employed, but *thus far (mark the words)* the data do not appear to me to warrant a continuance of their use. . . . "

Does it not seem from this that he had a premonition of the oncoming of insulin?

Numerous glandular or pseudoglandular preparations have been marketed with the claim that, as they contain the extract of the islet tissue, as well as (or without) the rest of the pancreas, they will supply what the diabetic lacks if properly taken. An enteric coated tablet has been marketed which is certainly more rational, but, if any of these have helped, except in that they supply what may be lacking in enzymatic secretion of the gland, it has not been proved; a few clinicians think that they have seen some benefit from these preparations when used as an adjunct to insulin therapy.

About thirty years ago, German investigators brought out several preparations which they thought would prove to be an oral hypoglycemic preparation of value, notably synthalin, neo-synthalin and glukhorment. In fact, so well were they received that Prof. Frank, of Breslau, under whose supervision synthalin was first produced, received the Nobel prize in medicine, in 1927, for his work along this line. Although there has been reported some similarity in effects of synthalin and glukhorment, in that the lowering of blood sugar with them is due to the guanidine or guanidine derivative contained therein, my personal experience is limited to the first-named preparation. My information is that neosynthalin, intended to be an improvement on the original product, in that it is less toxic, has turned out to be much less effective. The objection to the use of synthalin has been that, if continued over a long period of time, serious gastrointestinal disturbance might be produced. It is partly on this account and partly because of difficulty in obtaining it at that time that I did not use it more often. In my limited experience no gastrointestinal irritation was observed.

As to diet, briefly, I would like to state that the pre-insulin treatment of "gradual starvation treatment" of Allen in this country and Guelpa in Italy produced the best results in severe complicated cases, while Newburgh and Marsh in Ann Arbor were advocating high fat diets with less protein, in spite of danger of acidosis. Of course, diets have undergone drastic

changes since the advent of insulin, except for lowering calories for overweights.

The socalled "Banting era" (Joslin) was preceded by notable preliminary work by von Mering and Minkowski in Germany, Allen, Barron and others in this country before the assiduous work of Banting, assisted by Best and encouraged by McLeod and Collip in Canada brought forth the present boon to diabetics and revision of our therapy of diabetes. Today, we have the regular, unmodified insulin, zinc insulin crystals, NPH and PZI and Globin modifications, not to mention the latest advance in the three degrees of Lente insulin. There is some question as to whether or not the protamine in NPH and PZI might be harmful, from long continued use, in fostering advance of arteriosclerosis; to this question, I have been unable to get a satisfactory answer from manufacturers.

ORAL PREPARATIONS

Now, it has often been said "There is nothing new under the sun"! Following the steps of Frank and others in Germany, the fellow-countrymen there have been still working on an oral preparation to replace insulin and they claim great benefits from sulphonamide derivatives, of which we have been trying Carbutamide (BZ 55) and Tolbutamide (Orinase) in this country. Dr. Herold, Jr. and I have had extensive experience with both these products and I would like to report that we have had excellent results from the former and moderately satisfactory use of the latter. However, due to the fact that we have had some unfavorable side effects from Carbutamide and a number of such reported to Eli Lilly & Co., who produce this, causing them to suspend clinical trials with it, we have all but abandoned this and are using Tolbutamide practically altogether now. However, we still have some patients (I have two) who insist upon continuing to be supplied with BZ-55 and, when we let them have it, we caution them that they are using it at their own risk. Some of my cases, at present, with care as to diet, are getting along very nicely with the Orinase tablets and without insulin; others are still taking insulin, but have been able to reduce the dosage considerably.

As to how these preparations act, there has been no clear explanation, but it must be either (1) by stimulating the endogenous secretion of the Beta cells of the pancreas, or (2) by depressing the action of the Alpha cells, which secrete glucogon, the insulin antagonist, or (3) by depressing the glycogenic function of the liver, thereby lessening the glucose production in the body. Whether or not the mode of action will work permanent harm to the body remains to be seen.

Despite the ever-busy Germans, attempting to get us a substitute for insulin, the organic extract is still the standby, generally speaking, in diabetes, especially in complications. There are several things, relative to these tablets, to be worked out, just as the mode of insulin action has never been fully explained; nor has the percentage of storage of glycogen in muscles and other tissues been thoroughly ascertained. Soskin's work on importance of liver function in diabetes should be emphasized.

I shall close by quoting a conclusion in a paper read by me before the Southern Medical Association in 1936:

"I venture the prediction that not until we have found how to control the glycogenic function of the liver and, in addition, perhaps, how to regulate the natural secretion of insulin, will we have reached Utopia in diabetic therapy."

DISCUSSION

Dr. Frank W. Pickell (Baton Rouge): I certainly enjoyed this paper and congratulate Dr. Herold on his scholarly presentation.

Some months ago, I read one of Dr. Herold's earlier papers, written in the late twenties, mainly concerned with juvenile diabetes. His ideas of etiology and management were practically what they are today with the exception that we have better insulin today.

I am sure that his ideas then were considered somewhat radical but today, they are the commonplace. I think, Dr. Herold is not only a historian of diabetes, but also a prophet and his paper today, again looks into the future. I think papers like these really help us.

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DIABETES COMPLICATED BY SURGERY *

DANIEL W. HAYES, M. D.†
NEW ORLEANS

Bad risk—acidosis, coma, insulin shock, electrolyte problems, anesthesia reactions, wound infection, delayed healing are among the bleak thoughts which may flash through the mind of the surgeon suddenly confronted with a diabetic patient upon whom he may have to operate. These problems were commonplace prior to insulin use, but there has been an extraordinary reversal of the morbidity and mortality statistics in the past ten years. In that time, there has been a decrease in the mortality of 12 per cent or more to a mortality rate almost that of the general population. Therefore, we now withhold surgery from no one just because of diabetes.[1]

DIAGNOSIS OF DIABETES

Initially, let us spend a moment on the diagnosis of diabetes. It should be emphasized that the triad of polydypsia, polyuria, and polyphagia associated with loss of weight will not be present in many undiagnosed diabetics, nor will a urine specimen be positive for sugar in all diabetics preoperatively. Remember the high renal threshold for sugar, often 200 mgm. per cent or more, that exists in the elderly, nephrosclerotic patient. A preoperative blood sugar may be life-saving in such a case. It is never easy to diagnose postoperatively in a stuporous patient receiving intravenous glucose. Under stress of an acute infection or trauma, some "prediabetics" will have hyperglycemia and glycosuria. If the surgeon is always aware of the fact one out of 80 persons is a diabetic some needless morbidity and perhaps mortality could be avoided by careful preoperative examinations of all patients, especially the patients who are obese, or women with pruritus, or people with arteriosclerotic changes at an early age, or those patients with diabetic relatives.

PREOPERATIVE PREPARATION

If the patient is a diabetic, what then? If the surgery is elective, it is advisable to postpone the operation and allow adequate time for evaluation and preparation of the patient. This time varies from a few days to several weeks. The risk is increased in the presence of obesity, cardiovascular renal disease, and of course, in patients with poor control of diabetes. During this observation period, he should be brought into metabolic balance, not only for sugar but also for proteins, fats and electrolytes, by regulation of diet and adjustment of insulin dosage and type if necessary. Long-acting insulin, such as protamine zinc, should always be discontinued and an intermediate insulin, such as NPH or Lente, be substituted, since there is much difficulty with the day-to-day adjustment of PZI because of a two and a half to three day overlap of dosage, and nocturnal hypoglycemia is frequent at high dosage levels. Attention should be paid to protein and nitrogen balance, an important factor in wound healing. A new diabetic patient particularly should be placed in positive nitrogen balance. The fasting blood sugar ideally should be brought to a level between 120 to 150 mgm. per cent. Electrolytes usually are already in balance unless keto-acidosis is present, but must be checked. The vascular state of the patient must be evaluated. Since arteriosclerosis is a complication that appears early in the diabetic's life, all but the youngest should have electrocardiograms made preoperatively. Peripheral pulses must be palpated routinely, and a written record be made of the findings. Because of some increased susceptibility to tuberculosis, all diabetics should have chest x-rays done routinely before operation.

PREANESTHETIC MEDICATION

Preanesthetic medication deserves only a brief comment in that diabetics may take the same preoperative sedation indicated for the age and vascular state of

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any patient. It is wise not to oversedate the patient, since the subsequent respiratory depression may lead to hypoxia, especially in the elderly.

CHOICE OF ANESTHESIA

The choice of anesthesia is a somewhat more important consideration.[1, 3, 4] The anesthetic which is the safest is the one which interferes least with the hormonal mechanisms. In this regard, it is felt that ether is contraindicated because of its sympathicomimetic actions, causing hyperglycemia in addition to its usual tendency to cause nausea and vomiting. Cyclo-propane has little or no adverse effect on diabetics, nor does ethylene. However, in all possible cases, local, spinal or caudal anesthetics should be used. Intravenous pentothal is also well tolerated, though if the liver is diseased, the use of this anesthetic agent may be unwise.

EMERGENCY SURGERY

If emergency surgery is unavoidable, operation need not be delayed for merely the presence of hyperglycemia and glycosuria, but if acidosis is present several hours delay may mean a successful operation and a live patient. In this regard, one must recall Zachary Cope's admonition that "impending coma may cause a misdiagnosis of acute inflammation within the abdomen."[2] During these few hours serum electrolytes and sugar are always determined at the moment the patient is first seen. Crystalline insulin is given, often giving half of the dose intravenously and the other half subcutaneously. The initial dose varies as to the severity of the acidosis and the hyperglycemia. If it can be determined how long the patient has been ill and to what extent he has been out of control, the problem of dose will be easier. Forty to fifty units of crystalline (or regular) insulin initially will reduce the blood sugar considerably in three to five hours. In that time, the results of the chemistry studies will be available, and more insulin can be given if necessary. If the acidosis is mild and the patient is clinically not in a severe state a smaller dose of 15 to 20 units subcutaneously is advisable. Saline and electrolyte solutions are administered to rehydrate the patient. Blood is given as indicated, as are antibiotics.

If surgery is to be done in an acidotic patient without delay, as in certain traumatic and other life-saving procedures, one needs to give crystalline insulin in dosage ranging between 20 and 40 units intravenously initially to insure absorption, if the patient is severely ill. The insulin should NOT be mixed with other fluids in the flask. Five per cent glucose with saline is given during the surgery. When the patient is in the recovery room and thereafter, regular and frequent observation can be carried out with the administration of insulin, fluids, and electrolytes as indicated.

ELECTIVE SURGICAL MANAGEMENT

Diabetics going to surgery as scheduled cases should be the first early morning case, and a plea is made to surgeons and operating room personnel to make this priority a rule in handling such cases. Not only is the feeding problem made much easier, but postoperative blood sugars may be made by the regular laboratory technicians, and thus will be more accurate in most cases.

Hypoglycemia with its dangers, and ketosis with its serious implications, are to be avoided during and after surgery. Glycosuria and hyperglycemia are to be expected postoperatively. Therefore, the insulin dose preoperatively is to be determined with these factors in mind. A blood sugar should be drawn in every case before surgery.

The problem of insulin dosage may be handled in two different ways, the choice depending on the habits of the clinician and the severity of the diabetes. Insulin is omitted preoperatively completely by some, who prefer to control the patient entirely with crystalline insulin at stated intervals. This should always be the case in patients taking 20 units or less each day and, of course, those patients taking PZI should have been changed to an intermediate form, NPH, Globin or Lente, at

least three days prior to surgery. During surgery, 5 per cent glucose in water is used, and a small amount of crystalline insulin, usually about 15 units, is given subcutaneously or into the tubing for each 1000 cc. of fluid used. This is less than the usual amount needed to "cover" the 50 gm. of glucose administered. Never should insulin be added to and mixed in the flask, for its effect will be long delayed and almost worthless, as it drips in unit by unit. Rather it should be given all at once, for its effect in any case is not fully developed until three to six hours later.

Another way of handling this problem is to give half of the usual dose of the intermediate-acting insulin on call to surgery, then adding no additional crystalline insulin at any time during the operation, until at least twelve hours later. Five per cent glucose in water is administered during and after surgery.

POSTOPERATIVE DIABETIC CARE

Postoperatively in either case, the patient is observed and handled according to Woodyatt's six-hour management program. Electrolytes, combined with the glucose solutions and blood, are given as indicated and the problem is no different than in the nondiabetic patient. Blood sugars are often advisable late in the afternoon after surgery, and should be obtained routinely each morning thereafter, until the patient is well controlled.

The six-hour management program is based on the timing of crystalline insulin, so that there is peak effect of the insulin between four and six hours, with little overlapping of dose. It is a twenty-four hour control of the patient, not merely a daytime sixteen-hour schedule. Intravenous feedings are given at a rate of about 40 to 50 drops per minute, or 800 cc. in each six-hour period, so in twenty-four hours, 3,200 cc. of fluids may be given. Urine tests for sugar and acetone are obtained every six hours, or more often if desired. Crystalline insulin is administered subcutaneously (never in the flask) every six hours depending on the reaction of the urine for sugar. Usually, the ideal reaction is maintained at about 2 plus (¾ per cent) with a negative acetone test. If the urine becomes negative for sugar in the presence of adequate renal function, the insulin should be omitted entirely, since the danger of hypoglycemia is severe. If the urine reaction shows 4 plus glycosuria, give 20 units of crystalline insulin; if 3 plus give 15 units; if 2 plus give 10 units; and if 1 plus or a trace give 5 units. Blood sugars of 180 to 200 mg. per cent after surgery are satisfactory. This successful program of six-hour management should be continued until the patient is able to take normal oral nutrition.

For the patient who can eat postoperatively without nausea or vomiting, this will obviously be the ideal method of nutrition. Late afternoon feedings of small amounts of oral liquids, including tea, coffee, orange juice, ginger ale, gruel and thin oatmeal are given. Six-hour management then need not be continued longer, if the patient is able to eat soft foods and a full caloric program of diet can be planned with only one morning dose of intermediate insulin. Thereafter, urine sugar and acetone are estimated on a "q.i.d.", a.c., and bedtime basis.

SUMMARY

Accurate diagnosis of the unknown diabetic and meticulous care of the known diabetic make the risk of any surgery in a diabetic patient no greater than that of the usual patient. A period of preoperative observation and care to get the patient in perfect metabolic and electrolyte balance is essential in avoiding needless risk and morbidity.

Emergency surgery need never be delayed merely for diabetes, though if acidosis is present a few hours of preparation may be lifesaving.

Local, caudal, or spinal anesthetics are the ideal choices, but ethylene and cyclopropane may be used. Ether should not be used in a diabetic patient.

Six-hour management during and following surgery makes the fluid and elec-

trolyte problem much more easily handled. Crystalline (regular) insulin is administered every six hours around the clock, depending upon the amount of glycosuria present until the patient can tolerate oral fluids and food.

REFERENCES

1. Bortz, W. M., Bortz, E. L.: Surgery of the diabetic patient: Changing concepts, Am. J. Clin. Nutr. 3:494 (November-December), 1955.
2. Cope, Z. M.: The Early Diagnosis of the Acute Abdomen, 10th Edition London, New York, Oxford University Press 1951; 270 Pp.
3. Goodman, J. K.: Management of diabetes mellitus during ketoacidosis, acute infection and surgery, J.A.M.A. 159:831 (October 29), 1955.
4. Spence-Sales, Dorthey, Mehta, Mark: Some endocrine problems in anesthesia for emergency surgery, Medicine Illustrated, 9:639 (October), 1955.

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COMPLICATIONS OF SUBMUCOUS RESECTION *

RALPH H. RIGGS, M. D.
SHREVEPORT

INTRODUCTION

The operation of submucous resection of the nasal septum has been developed and refined many times since its original description, in 1882, by Ingals.[1] To Freer[2] probably belongs the credit for the first great scientific presentation that popularized the procedure at the turn of the century and led to its wide acceptance. However, during the past decade, because of the complications that may result from this operation, its merits have been subject to much questioning. Although by this procedure it is possible to correct a considerable number of pathologic septal conditions, many such abnormalities of the caudal end of the septum and along the dorsum cannot be as satisfactorily excised.

During the first forty years of the present century many valuable observations were made on the anatomy of the nose, and many technical improvements in submucous resection were suggested. In 1946, Cottle and Loring[3] described a technic for correction of nasal abnormalities based upon surgical and physiologic principles. Since then Cottle[4-6] has repeatedly called attention to the physiology of the septum and vestibule, and the importance of performing other rhinologic procedures along with the septal operation if normal nasal physiology is to be restored. Only with knowledge of the anatomy and physiology of the nose[7-10] can one recognize, help prevent, and correct the numerous complications that may result from submucous resection. Among these are sagging or saddling of the cartilaginous vault, retraction of the columella, ballooning of the upper lateral or lobular cartilages, widening of the base of the nose and lobule, depression or flattening of the nasal tip, atrophy of the septal mucosa, adhesions between the septum and lateral nasal wall, perforations, and psychologic changes due to nasal deformity or disturbed respiratory function.

ETIOLOGY

These complications may be classified etiologically into those due to (1) loss of support, (2) scar tissue, (3) loss of support and scar tissue, and (4) surgical sequelae. *Loss of support* is brought about by "total" severance of the continuity. The cartilaginous and bony elements of the septum are involved and support of the dorsum from the cephalic limits to the caudal end of the septum is interrupted. This does not necessarily mean that all the cartilage and bone have been removed. In fact, a longitudinal cut in the septum, without removal of any portion of it, may interfere with support. This is often encountered in an injured septum and results in a deformity of the external nose and some of the listed complications.

The amount of *scar tissue* necessary to produce complications depends upon the patient, the neighboring structures, the extent of the surgical procedure, the propensity of the tissues and the steps that are taken to minimize or control formation of scar tissue. This was thoroughly discussed by Cottle,[4, 5] in 1948, and need not be considered further here.

The commonest *surgical sequelae* probably follows the operative injury to the mucopericondrium and mucoperiosteum. Another sequelae, perforation of the sep-

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tum immediately after operation, occurs rarely with present technics but is still an important possibility.

COMPLICATIONS

Although each complication will be considered here as a separate entity, this does not mean that these complications usually are encountered separately. In fact, it is more likely that several will be found in the same patient. Also, in this discussion it is assumed that none of these complications were present preoperatively.

Sagging of the cartilaginous vault is the most obvious sequelae of submucous resection. It may range from a slight deformity to complete saddling. This complication may be immediate (occurring during the operation) or latent (as a late result of injury, operation, or both). If it occurs during the operation, it is due to lack or removal of support, usually the septum. However, abnormalities in the structure of the upper lateral cartilages and unusual conditions of the skin and subcutaneous tissue, especially if scar tissue is present, can also be a factor in support. If this complication is latent, it is probably due to scar tissue, although the support was probably weakened at the time of operation.

Retraction of the columella usually is not seen until some time after the operation even though it is latent and can be demonstrated clinically. It is caused by loss of the caudal end of the septum or nasal spine, or both. This may or may not be combined with injury to component parts of the columella.

Ballooning of the upper lateral or lobular cartilages. After submucous resection of the nasal septum, scar tissue may pull the cartilaginous dorsum toward the nasal cavities. This can increase the angle formed by these cartilages and the septum and thereby create a valvular abnormality. The lobular cartilages can be affected in the same way. *Ballooning of the lobular cartilages* is usually more noticeable in patients in whom the caudal end of the septum has been removed.

Widening of the base of the nose is always a real threat in the presence of loss of support of the nasal septum or nasal spine and the possibility of scar tissue pull. Cottle [3] cautioned that every septum operation with possible "totality" requires prophylactic repair of the wide nose, since widening is apt to occur after a submucous operation.

Depression of the tip with an acute nasolabial angle can be due to too generous removal of the septum, especially along the anterior inferior portion, caudal end and spine. *Flattening of the tip, flaring of the wings of the lobular cartilage,* or both, may result from a pulling down (backwards) of the medial crura and dome and is due to cicatrization.

Atrophy of the septal mucosa may be caused from injury to the mucopericondrium and mucoperiosteum and can result in a flabby septal membrane, crusting, frequent bleeding and finally perforation. Atrophy of the septal mucosa may or may not be associated with general nasal atrophy (atrophic rhinitis). *Adhesions* between the septal mucosa and lateral nasal wall, of course, are due to scar tissue forming between two injured areas.

Psychologic changes may be due to nasal deformity or disturbed respiratory function. The patient with disturbed respiratory function may complain of poor sleep, fatigue, allergic manifestations, nasal insufficiency, head pains, referred pains and symptoms referable to the chest.

The possibility of the development of such numerous and serious complications makes one reevaluate submucous resection operations and try to determine what can be done to prevent such complications. There is no doubt that the procedure is of great value in indicated cases, but particular care should be exercised to prevent development of complications. It should be possible to remove all the cartilaginous and bony septum, if necessary.[11] The septum should then be reconstructed to restore normal anatomic and physiologic relationships. Other rhinologic procedures should be performed along with the septal operation, if indicated and if

necessary to achieve a more desirable end result.

It should be remembered that in the patient whose external nose is malaligned, twisted or deformed, it is impossible to obtain a good functional result without some attention to the external pyramid (Fig. 1). Pathologic alterations of the upper lateral cartilages and lobule should be corrected at the time of the restitution procedure. Malformation, poor development or injury to the premaxillary area may cause underdevelopment or absence of the nasal spine (Fig. 2). This may be reconstructed to help produce a better functioning respiratory organ. In addition, pieces of cartilage (autogenous or isogenous) placed in the necessary region will help prevent deformities and also aid in the process of healing (Fig 3.).

SNMMARY

Submucous resection is a valuable procedure in indicated cases but it should be performed only by those well versed in the anatomy, physiology, and mechanics of this region, if serious complications are to be prevented. Among these are sagging or saddling of the cartilaginous vault, retraction of the columella, ballooning of the upper lateral or lobular cartilages, flattening of the nose with or without depression of the tip, adhesions between the septal mucosa and lateral nasal wall, widening of the base of the nose, atrophy of the septal mucosa, perforations, injury to the mucoperichondrium and mucoperiosteum, and psychologic changes. These complications are due to loss of support, development of scar tissue, or a combination of these. It should be possible to remove and restore all the cartilaginous and bony septum, if necessary, and preserve normal anatomic and physiologic relationships.

REFERENCES

1. Ingals, E. F.: Deflection of the septum narium, Arch. Laryng., 3:291 (Oct. 1) 1882.

2. Freer, O. T.: The correction of deflections of the nasal septum, with a minimum of traumatism, J.A.M.A., 38:636 (Mar. 8) 1902.

3. Cottle, M. H. and Loring, R. M.: Corrective surgery of the external nasal pyramid and the nasal septum for

Figure 1.—(Left) Preoperative photograph of deformities of the bony and cartilaginous vaults and lobule, and an obstructing septum.

(Right) Photograph of patient after complete removal of septum with reconstruction, realignment of external pyramid, and operation on the upper lateral cartilages and lobule.

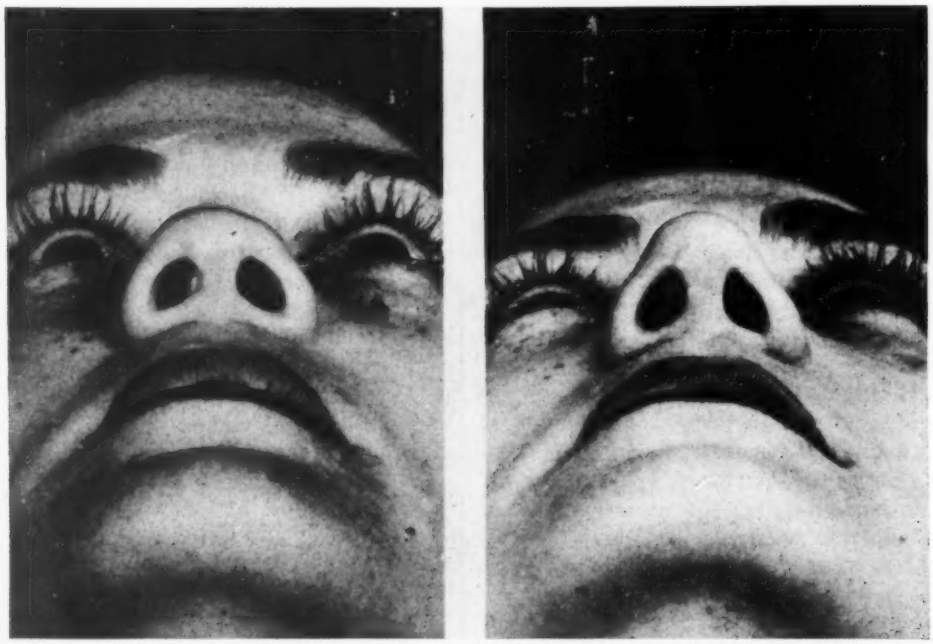

Figure 2.—(Left) Preoperative photograph of injured external nose, lobule and caudal displacement of septum.

(Right) Photograph after complete septal operation, with reconstruction, and procedures on external pyramid and lobule.

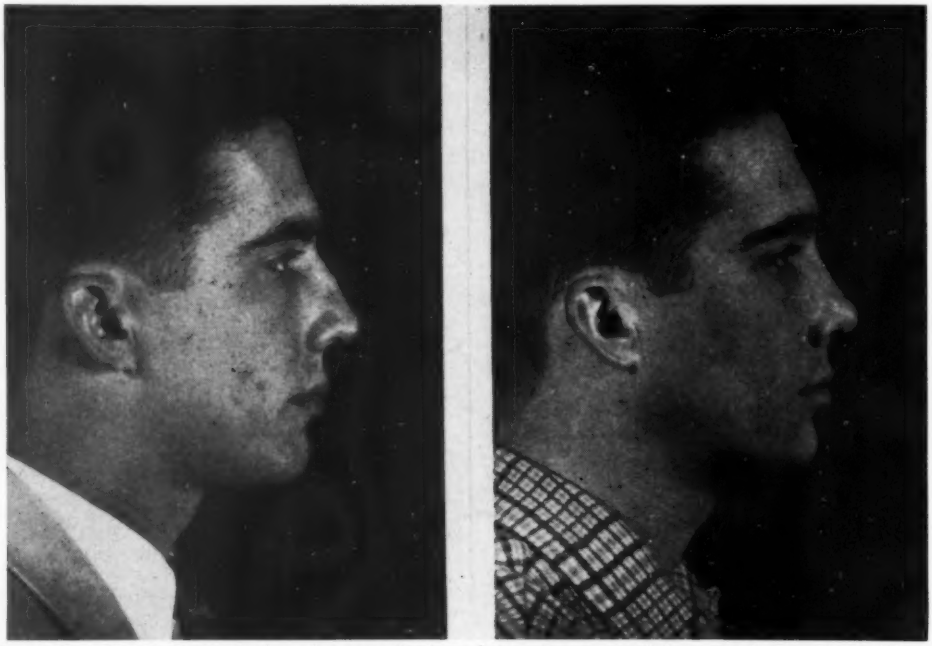

Figure 3.—(Left) Preoperative photograph of an injured nose requiring septal operation with reconstruction and procedures on the external nose, including many small cartilaginous grafts over cartilaginous dorsum.

(Right) Postoperative photograph.

restoration of normal physiology, Illinois M. J., 90:119 (Aug.) 1946.

4. Cottle, M. H. and Loring, R. M.: Newer concepts of septum surgery: present status, Eye, Ear, Nose & Throat Month., 27:403 (Sept.) 1948.

5. Cottle, M. H. and Loring, R. M.: Surgery of the nasal septum: New operative procedures and indications, Ann. Otol., Rhin. & Laryng., 57:705 (Sept.) 1948.

6. Cottle, M. H.: Modified nasal septum operation, Eye, Ear, Nose & Throat Month., 29:480 (Sept.) 1950.

7. Williams, R. I.: Functions of the nasal septum as related to septal reconstructive surgery, Laryngoscope, 63:212 (March) 1953.

8. Riggs, R. H.: The saddle nose, South. M. J., 44:188 (March) 1951.

9. Riggs, R. H.: The relation of septum reconstruction to external deformities of the nose, Laryngoscope, 63:293 (April) 1953.

10. Cottle, M. H.: The structure and function of the nasal vestibule, A.M.A. Arch. Otolaryng., 62:173 (Aug.) 1955.

11. Tucker, C. A.: Total removal of nasal septum, Presented before the meeting of the American Rhinologic Society, Oct. 10, 1956.

DISCUSSION

Dr. F. W. Raggio, Jr. (Lake Charles) "But don't let him operate on you!" I am sure that most all of you have either said those words to patients or have had patients say them to you. I know that I have had patients referred to me with this admonition—don't let him operate on you. I bring this up to illustrate the low point to which rhinologic surgery has descended. This was during the time which I call the "Dark Ages" of Rhinology, prior to 1945. This was before Dr. Cottle had begun to spread his gospel of septum and pyramid surgery and before Dr. Hansel's allergic teachings were widespread.

There are, in my opinion, two reasons for this low ebb in rhinologic surgery. First, is the inadequacy of the classical Killian type of submucous resection. This point was adequately covered by Dr. Riggs. Second, was our lack of knowledge concerning the diagnosis and treatment of nasal allergies.

This concept of nasal surgery, which Dr. Riggs had described, has opened up new horizons for the rhinologic surgeon. First, it enables him to do much more extensive procedures on the septum itself, especially the important caudal end, which we could not touch in the Killian type of operation. Second, it enables him to deal with the abnormalities of the external nasal pyramid, including the upper and lower lateral cartilages at the same time. Thirdly, and this was the point of Dr. Riggs splendid presentation, that complications can be prevented and corrected at the time of surgery.

Along with broadened horizons in rhinologic surgery has come an increased understanding of the nose from a physiologic standpoint. The knowledge of the "valve area" and the baffles and their effect on the air currents has enabled us to improve our rhinologic surgery physiologically, as well as anatomically. As mentioned by Dr. Riggs, Dr. Maurice Cottle is largely responsible for this new concept; the knowledge of which Dr. Riggs and others throughout the country have generously shared with the specialty as a whole.

But don't let him operate on you! These words need not be said when referring your patient to a rhinologist well versed in present day Rhinology. We are now out of the Dark Ages.

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PRIMARY SYSTEMIC AMYLOIDOSIS: CUTANEOUS MANIFESTATIONS A CASE REPORT *

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The following case is presented as an example of an interesting and unusual condition. Only 75 cases of this rare disorder were recorded up to 1955.[1]

The skin as well as the internal organs may be the site of deposits of amyloid. This is a homogeneous degenerative substance of uncertain and apparently inconstant chemical composition. Amyloid, meaning starchlike, does stain with iodine. However, with iodine it turns a mahogany brown. Upon addition of sulfuric acid, a dark blue color forms, hence the designation of the substance as amyloid by Virchow.[2]

Amyloid is a complex protein characterized physically by its hyaline, structureless nature. It is known, histochemically, for its metachromasia, in that it takes a reddish color with such purplish dyes as gentian violet and crystal violet. This is evidence for a histochemical interaction between amyloid and these dyes. Mere adsorption of dyes does not ordinarily result in color changes of the dyes. Amyloid deposits are also known for their ability to absorb Congo red from the circulation.[3-5]

Secondary amyloidosis classically is related to protracted suppuration. Empy-

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ema, lung abscess, and osteomyelitis were notable offenders, however, today's antibiotics have changed this picture. Secondary amyloidosis also occurs in association with plasmocytosis, Hodgkin's disease and chronic ulcerative colitis. The incidence of amyloidosis in multiple myeloma varies in different series, averaging 15 to 20 per cent. In secondary systemic amyloidosis the involvement is usually of the parenchymatous organs; in particular, the spleen, kidneys, liver, and adrenal cortex are involved. Amyloid nephrosis is a frequent cause of death in leprosy. The skin is little and rather uncharacteristically involved in secondary systemic amyloidosis.

In primary systemic amyloidosis, amyloid is deposited throughout the body. The blood vessels, connective tissues and muscles bear the brunt of the pathology in this form. Almost one-half of these patients have cutaneous manifestations. Purpura is the commonest skin lesion in primary systemic amyloidosis. A set of symptoms and signs occurs here with sufficient regularity to distinguish this form dermatologically.[6] These signs and symptoms are: (1) a characteristic cutaneous eruption, (2) purpura and ecchymoses, (3) glossitis and macroglossia may be present, (4) pains and aches in the musculature, and (5) alopecia may also be present.

CASE REPORT

A 63 year old negro male was admitted to the dermatology ward of Charity Hospital in New Orleans on April 19, 1956. His complaints were redness around the eyes and "a weak skin that breaks easy".

Eighteen months prior to admission, he noted a reddish discoloration about both eyes. He was otherwise asymptomatic at this time.

One year prior to admission he had to give up his work as a masonite cutter which he had done for years. He found that his arms tired during his work day. In the morning he felt fine, but his arms in particular soon tired; his fatigue then became general. He was forced to leave work earlier and earlier, finally having to give up his employment. About this time he noted his skin becoming what he considered "weak and easily broken". This was initially noticed when, following urination, the required genital manipulation resulted in reddening and bruises in that area. About this time also, the skin over his knees became reddened and bruised in appearance. He further noted that any slight rubbing or injury at almost any location would result in these reddenings and bruises in the area.

About six months prior to his admission, he noted loss of hair from his axillae and pubic areas. These areas were otherwise asymptomatic. In addition, he admitted mild dyspnea upon climbing stairs. This was believed somewhat compatible with his sixty-three years. He did have nocturia times two.

Past history and family history were noncontributory. He was a man of exemplary habits, a nonsmoking teetotaler.

Physical examination: About both eyes were grouped shiny, smooth, firm spherical papules, of a reddish waxy color. Complete hair loss in both axillae and pubic areas had occurred (Figure 1). Scalp hair was thinned out but apparently this was physiologic. There was also some eyebrow thinning. Ecchymotic areas were present over his inguinal and perianal regions (Figure 2). Ecchymosis were most pronounced however, over both knee areas. Closer inspection revealed smaller scattered ecchymoses over various body areas. Two iatrogenic ecchymoses were present,

Figure 1. Showing papules about the right eye, and axillary alopecia. The darkened area in the axilla is an intracutaneous Congo red test, positive after three weeks.

one where adhesive tape was placed and removed from over his sternal area following bone marrow studies, the other over the upper eyelid which was grasped to insert eyedrops prior to ophthalmologic examination. This gentle maneuver ecchymosed his entire upper lid.

Figure 2. Showing circumanal ecchymoses.

His tongue appeared mildly hypertrophic. X-rays of his chest, skull, long bones, and gastrointestinal tract were negative. Rumpel-Leede tests were negative on two occasions. Medical consultation and presentation to the Tulane medical conference of Charity Hospital substantiated the opinion of primary systemic amyloidosis. An electrocardiagram was within normal limits. Ophthalmologic consultation was essentially negative.

Laboratory: Liver and kidney investigations were without abnormal findings. A fasting blood sugar was 78 mgm. per cent. Serum proteins were within normal limits. No cryoglobulins were found. The S.T.S. was negative. Two hematologic consultations were obtained. The first was concerned with the peripheral blood and coagulation studies and was reported as a very mild normochromic normocytic anemia with a minimal lymphocytosis. Sickle cell prep was negative. No coagulation abnormalities were present. The second hematologic consultation was concerning a bone marrow study, the report was mild hypoplastic bone marrow. Urine studies showed a two plus albumin on one occasion. Several other specimens were negative. Tests for Bence Jones protein were negative on two occasions. A Congo red test was performed. The intravenous method of Benhold [5] was first considered, but for safety reasons [7] and probably because, as dermatologists, we prefer to see what is going on whenever possible, we selected the suggested method of Nomland,[3] which is an intracutaneous injection of Congo red. We used 0.1 cc. of a 1 per cent solution and this delineated well the inguinal and axillary amyloid areas. Positive test areas remained as deeply pink stains for the five weeks we were able to observe this patient. Skin discoloration from intracutaneous Congo red was gone from the nonamyloid regions after a few days. Positive Congo red skin tests resemble merthiolate * stains and were mistaken for them. It is suggested that when used, this test be given in areas showing amyloid histologically and also given in nonamyloid areas as controls. Our pathology department advised us that to biopsy this Congo red test area would be to no avail for histological study because of the small amount of dye that might be in the microscopic section.

Pathology reports: Positive findings in the right axilla, the gingiva [8] and the outer lower right eyelid. The scalp was negative for amyloid histologically. The pubis showed amyloid deposits around the skin appendages, particularly the hair follicles.

* Trade Mark

COMMENTS

According to Dahlin,[9] cutaneous manifestations occur in primary systemic amyloidosis in 50 per cent of the cases. Macroglossia occurs in 50 per cent of the cases. There was only slight indication here of its presence. Multiple myeloma is frequently found in association with amyloidosis. It was not present here.

The skin findings in secondary amyloidosis are almost nonexistent, and atypical when present.

The skin findings in primary systemic amyloidosis herein presented, we believe to be characteristic.

SUMMARY

A case of primary systemic amyloidosis is presented. We believe the dermatologic associations, when seen, are characteristic of this rare condition.

REFERENCES

1. Cecil, R. L., and Loeb, R .F.: Textbook of Medicine, Philadelphia, W. B. Saunders Co., 1955, p. 718.

2. Virchow, R. L. K.: Cellular Pathology Based on Physiology and Pathological History, New York, Robert DeWitt, 1860, p. 409-437.

3. Nomland, R.: Localized (lichen) amyloidosis of the skin, A.M.A. Arch. Dermat. & Syph., 33:85, 1936.

4. Harman et al.: A.M.A. Arch. Int. Med., 70:416, 1942.

5. Benhold, H.: Ueber die Ausheidung Intravenos Einverleibten Kongorotes bei den Verschiedensten Erkrankungen Insbesondere bei Amyloidosis, Deutch Arch. f. Klin. Med., 142:32, 1923.

6. Michelson, H. E., and Lynch, F. W.: Systematized amyloidosis of the skin and muscles. Arch. Derm. & Syph. 29:805 (June) 1934.

7. Selikoff, I., and Berstein, I. J.: Systemic reaction to the intravenous administration of Congo red, Quart. Bull. Seaview Hospital, 8:131, 1946.

8. Selikoff, I., and Robitzer, E.: Gingival biopsy for the diagnosis of generalized amyloidosis, Am. J. Path., 23:1099, 1943.

9. Dahlin, D. C.: Primary amyloidosis with a report of six cases, Am. J. Path., 25:105, 1949.

DISCUSSION

Dr. Vincent Derbes (New Orleans): This interesting case report stimulates discussion of several points. In the first place, their wisdom in avoiding an intravenous Congo red test is attested to by an unfortunate medical experience in Minnesota. In this instance a man with this condition was subjected to an intravenous Congo red test. The test was positive in the sense that the dye-stuff was taken up by the patient's skin. The generalized vivid red color has now persisted for several years.

Again, the authors point out that reduced incidence of secondary amyloidosis is probably due to the effectiveness of antibiotic treatment of chronic suppurative processes. On the other hand, in conditions in which the response to such treatment is slower, amyloidosis continues. Thus, in the past year we have seen at Charity Hospital a half dozen new cases of leprosy, the majority with amyloidosis.

Numerous theories have been advanced on the production of amyloid in the organism. The most widely accepted theory today is that the precipitation of amyloid is the result of an antigen-antibody reaction. This theory receives support from the very frequent occurrence of amyloidosis in hyperimmunized horses used for the production of tetanus antitoxin. It has also been possible to induce amyloidosis in experimental animals by the injection of pus, sodium caseinate, Freund's adjuvant and numerous other substances. A feature common to all experimental studies, as well as to many human instances, is the increase in the absolute amount of blood globulins. Thus chronic hyperglobulinemia seems to be an important factor in the production of secondary amyloidosis. In the type of amyloidosis under discussion, this hyperglobulinemia need not be present.

There is no effective treatment for this condition. Experimental work in animals indicates that corticoids are injurious and suggests that, at least in the secondary type, thyroid substance is beneficial.

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THE DIAGNOSIS AND CONSERVATIVE TREATMENT OF LOW BACK PAIN

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In almost all specialties and certainly in the general practice of medicine, backache is a common complaint heard many times daily. The attitude that very little can be done for backache is still held by many laymen and even some in the medical profession. Such a pessimistic attitude is not justified if an accurate diagnosis is combined with treatment that is tailored for the individual case.

DIAGNOSIS

In the diagnosis of back pain we follow the same procedure as we should do for all patients, and this consists of a detailed history, physical examination, x-ray studies, and if necessary, laboratory studies. We must keep in mind that back pain can be caused by nonorthopedic as well as orthopedic conditions. One method of classification of orthopedic backaches is to place them into one of the following four categories; mechanical, congenital, inflammatory, and neoplastic. The nonorthopedic conditions include genitourinary, gynecological, visceral, and systemic abnormalities. This discussion will be limited to certain aspects in the diagnosis and conservative treatment of low back pain arising from orthopedic conditions. Most of the causes for backache are on a mechanical basis and include such conditions as sprains, strains, postural back pain, and protruded or degenerated intervertebral discs. A lumbosacral pain is ordinarily a result of an injury. The onset can be acute and painful as a result of a severe injury. On the other hand the disability can be just as great if it occurs while stooping over to pick up a pencil from off the floor. The history may suggest a chronic strain due to poor postural attitudes such as from long hours over a workbench. Careful explanation of the underlying cause to the patient and employer is of more benefit than a "muscle relaxant" prescription after a cursory examination.

The protruded disc is the most common cause of low back pain with sciatic radiation. Sciatic radiation must be one of the complaints before such a diagnosis is seriously entertained. Intermittent low back pain for several months or years before

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sciatica is the history in about half of the cases. It is important not to consider that all cases with sciatic radiation are caused by protruded discs. Sciatic radiation can occur from nerve root irritation secondary to narrowing of the foraminal exit or from bony spurs or from inflammatory edema of the nerve roots from a sprain.

A patient with nerve root irritation or compression will usually relate that the radiating pain is brought on by certain back motions or from coughing or sneezing. Localization of the lesion may be suspected from hearing the patient describe the distribution of the radiating pain. Almost 95 per cent of the disc ruptures occur either at the fourth or fifth lumbar interspaces. Lumbosacral disc protrusion compresses the first sacral nerve root more than any of the other nerve roots. This may result in loss of the ankle reflex and decrease of sensation along the lateral aspect of the foot and leg. Calf atrophy is present if compression is not of fairly recent onset.

The disc between the fourth and fifth lumbar vertebrae usually compresses or irritates the fifth lumbar nerve root. There may or may not be reflex changes in the knee or ankle. There is frequently a varying degree of sensory loss in the first and second toes and dorsum of the foot. Weakness of dorsiflexion of the great toe is not uncommon.

Some authorities believe that lesions of the intervertebral discs are the basis of practically all back pain, while others say that there are varied causes. One may ask how can there be such a variance of ideas among experienced writers. It is true that the normal disc is very rarely the site of back pain, while there is increasing evidence to show that degenerating lower lumbar discs are common and result in a predisposition for unexpected or unusual strain. Certainly not all degenerated discs cause symptoms. Microscopic degeneration of intervertebral discs begins routinely in early life to a variable degree, depending on congenital and environmental influences. It is true that the protruding disc practically always occurs at the site of a degenerative disc, except possibly from acute trauma displacing a normal disc. A degenerated disc is often the site of instability and thus any form of stress, acute or chronic, is apt to result in back strain. The numerous articles on this subject of disc degeneration emphasize the importance, frequency and lack of entirely satisfactory explanation.

In patients with developmental or congenital defects as basis for back pain, the symptoms usually begin in early adulthood and the pain is made worse by activity and relieved with rest. A family history may denote similar difficulties in the past. Congenital anomalies such as transitional vertebra, asymmetrical facets, hemivertebra, spina bifida occulta, and spondylolisthesis are all fairly common. These conditions predispose to back pain because of impaired ligamentous support or attachment.

Inflammatory causes for back pain will not be discussed but include such conditions as Marie-Strumpell arthritis and tuberculosis. These conditions may be missed if not considered as possibilities or if the sacroiliac joints are not checked carefully.

Neoplastic involvement should also be considered and may be either a localized process or a generalized metastatic involvement. A benign lesion such as osteoid osteoma can give rather severe pain without much in the way of physical findings. Many of the neoplasms are insidious at onset but pain may be steadily progressive and unrelated to activity. A detailed discussion of the various neoplasms will not be attempted in this report. The physician should not forget to repeat films occasionally if the patient is not responding to treatment. In this way early lesions or infections may be diagnosed.

Several causes of backache mentioned in the earlier literature are now considered to be unusual causes of back pain. Among these are sacroiliac strain or sprain, hypertrophy of ligamentum flavum and pyriformis spasm. Fibrositis is

not a well defined entity and not as commonly diagnosed as in previous years.

Physical examination should be carried out with full exposure of the entire back and lower extremities. Observation of the patient as he walks into the office and prepares for examination is very helpful. The general posture is noted and also any deviation of the spine, such as a list, scoliosis, increased lordosis or flattening of the normal lumbar curve. A routine procedure for examination will help prevent overlooking certain parts of the examination. While observing the patient's back he is asked to move through various motions including flexion, extension, lateral bending and rotation. The examiner notes and records the degree of restriction, muscle spasm and tenderness. Some idea is obtained of the patient's pain threshold and how much he is trying to impress the examiner with his disability. The patient is then placed on the examining table in the supine position and the straight leg raising tests are carried out, as well as other leg tests that have been described. The straight leg test is probably the most valuable of these tests. A record is made of the location of the pain on raising the lower extremity with the knee extended. Also the number of degrees is noted at which the pain occurs, keeping in mind that tight hamstrings will give normally restricted straight leg raising tests with a sensation of tightness in the back of the thigh. Measurements are then carried out for leg lengths and circumference of the thigh and calf. A routine sensory examination with a pin is considered important. Examination is made to test the power of dorsi flexion of the big toe and ankle. The patient is then examined in the prone position. Palpation is made at the sciatic notch and the posterior thigh for sciatic tenderness. The knee and ankle reflexes are tested. Occasionally palpation of the back will reproduce the patient's pain and this may be helpful localizing any abnormality.

Roentgenological studies of superior quality are essential in diagnosing certain conditions and ruling out other diagnoses. The films are repeated if they are unsatisfactory. The AP, lateral and oblique positions, as well as a spot lateral of the lumbosacral joint are considered routine in most instances. One should not forget to check the sacro-iliac joints on the x-ray films in order to rule out such diagnoses as Marie-Strumpell or other infectious lesions. An old compression fracture of the dorsal spine may result in compensatory increase in the lumbosacral angle causing low back symptoms.

Laboratory studies are helpful in certain instances such as in multiple myeloma, infectious processes, Paget's disease, or other conditions causing low back pain.

TREATMENT

The treatment to be described in this report will be intended for the back pain arising on a mechanical or developmental basis, which includes the large majority of backaches. In general, the plan of treatment in most cases is to overcome the acute pain by rest and immobilization and then to gradually ambulate the patient without neglecting to improve muscle tone and posture in order to keep recurrences at a minimum. The underlying cause of each case should, of course, be determined and corrected as far as possible. One of the most common reasons for ineffective relief is the failure to keep the patient on bedrest long enough to overcome the acute pain before ambulating and discharging him back to work. The lack of follow-up in the management of the patient may predispose to recurrent episodes. These faults commonly stem from the patient's not carrying out instructions or from his failure to understand the purpose of the treatment. It is the doctor's responsibility to repeat instructions often enough for the patient to have a thorough understanding of the

program to be carried out.

On deciding to treat a patient for back pain, decision must then be made whether to treat him on an ambulatory basis, or on bedrest either at home or in the hospital. The severity of each case should decide which method to advise. The less severe and acute case can often be managed on an ambulatory basis, but in the more acute case bedrest at home for a period of two or three weeks may be carried out. If the patient has difficulty in walking, the recommended treatment is bedrest in traction for two weeks or until relief is obtained. In most cases physiotherapy consisting of diathermy and massage are beneficial, if there is muscle spasm. A corset support is frequently very helpful and in some cases quite important until the patient is able to strengthen the proper muscles sufficiently to carry on without support. Other methods of support such as strapping or bracing can also be utilized. In all cases a graduated program of exercises is recommended in order to overcome any contractures and improve the tone of the supportive muscles. These exercises must be postponed until most of the acute tenderness and muscle spasm have subsided. The most effective way to overcome the acute pain and muscle spasm is to place the patient on complete bedrest in the hospital in a Gatch type of bed in the semi-Fowler's position. Most patients will be more comfortable in this position than flat in bed. The Fowler's position overcomes the lordosis thus opening up the posterior part of the lumbosacral joint both at the foramen, which contains the nerve root, and the posterior part of the disc space. It takes the stress off of the facets and corrects any so-called settling or subluxation. The hospitalized patient is also placed in pelvic corset traction with from seven to ten pounds on each side. The pelvic traction helps to enforce complete bedrest, as well as relieving the back pain to a considerable degree. Sedation is given as required. The mattress should be moderately firm and nonsagging. Bedboards are placed under the mattress. The response is variable and an average of two weeks in traction is required. In some cases a lumbosacral support is fitted prior to getting up. Ambulation is very gradual to prevent recurrence until the muscles are strengthened. Postural exercises are advised in many cases. The exercises are of particular value in those cases with poor abdominal muscle tone and excessive lumbar lordosis, as so often seen in postural or mechanical type of back pain. The regular performance of a series of exercises as described by Williams will assist in flattening out the lumbar spine by strengthening the abdominal and gluteus maximus muscles and stretching of the contracted paravertebral muscles. A weight reduction program is of utmost importance if there is obesity. Recurrent strain may be avoided by telling the patient to avoid lifting heavy weights, to bend at the knees rather than at the low back region, to avoid hyperextending the lumbar spine as when carrying a weight at above the shoulder level, to avoid sleeping face down since this increases the lordosis, to relieve the strain from the low back region when standing by placing one foot on a small foot stool, and to avoid long automobile trips. As stated before, the majority of patients with low back pain and sciatica will respond to this conservative treatment.

One must keep in mind that continued conservative treatment in the face of progressive or continued neurological deficit is not justified and will likely result in an unsatisfactory result. The decision as to when to operate varies with each case depending on the number of previous attacks, degree of neurological deficit, extent of improvement on conservative treatment, age of patient, the degree of functional overlay, plus many other considerations.

Myelography is performed after decision is made for surgery. It is used to assist in localization and thus possibly

eliminate the need to explore two interspaces.

Manipulation of the back has a very limited place in the treatment of low back conditions. In certain carefully selected cases of an acute "catch" in the back without sciatic radiation and without x-ray abnormalities, manipulation carefully performed may result in dramatic relief of pain. An attempt should be made to correct any predisposing conditions following the manipulation.

Occasionally a patient will be found to have a localized painful or so-called trigger point in the superficial fascial or aponeurotic tissues. Steindler has shown that local anesthetization of this trigger point may result in some lasting benefit from the referred type of pain. If the pain recurs after a few injections, then excision of the trigger point lesion can be performed.

CONCLUSION

An attempt has been made to minimize the pessimistic attitude toward back pain in general. Not all back pain with sciatica is caused by protruded disc material.

The examiner must analyze a carefully taken history, a thorough physical examination along with sufficient x-ray and laboratory studies in order to adequately diagnose the numerous causes of low back pain.

The cooperation of the patient is essential in order to obtain the maximum benefit from a plan of treatment that may in some instances extend over a prolonged period of time.

REFERENCES

Williams, P. C.: Lesions of the lumbosacral spine: II Chronic traumatic (postural) destruction of the lumbosacral disc, J. Bone and Joint Surg., 19:690, 1937.

Mixter, W. J., and Barr, J. S.: Rupture of the intervertebral disc with involvement of the spinal canal, New England J. Med., 211:210, 1934.

Breck, L. W., and Basom, W. C.: Flexion treatment for low back pain: Indications, outline of conservative management and new spine fusion procedure, J. Bone & Joint Surg., 25:58, 1943.

PHENYLBUTAZONE (BUTAZOLIDIN) IN THE TREATMENT OF THROMBOPHLEBITIS; A PRELIMINARY REPORT *

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In the practice of Obstetrics and Gynecology at Charity Hospital of Louisiana in New Orleans, there are a fair number of patients exhibiting thrombophlebitic processes. Thrombophlebitis may arise, in patients with preexisting lower extremity varicosities associated with pregnancy or gynecologic pathology, as a retrograde extension of suppurative pelvic thrombophlebitis, as a sequela to the intravenous administration of fluids or medications, and spontaneously in postoperative, postpartal, or debilitated patients.

The overall management of treatment in patients with thrombophlebitis encompasses three general objectives: the prevention of embolization; the localization of the disease process; and the diminution of pain, inflammation and edema. To fulfill these goals, several methods of management have been devised.

The utilization of vena cava and bilateral ovarian vein ligation has certainly controlled embolization and localized the disease process in suppurative pelvic thrombophlebitis.[1-3] Ligation of other veins, i.e. iliac, femoral, or saphenous, have accomplished the same purpose in more distal localized phlebothrombosis or thrombophlebitis. Lumbar sympathetic blocks have proven beneficial in increasing the blood supply to the lower extremities in cases

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of thrombophlebitis by reducing vasospasm, in turn relieving a part of the pain and aiding in the reduction of edema. Antibiotics have played a role in the reduction of perivascular and intravascular inflammatory processes in those cases of an infectious etiology. They are, however, of little or no value in thrombophlebitis produced by chemical irritation of the endothelium. Anticoagulants have been used prophylactically in selected cases in an attempt to control embolization.

In the treatment of thrombophlebitis of the superficial veins, no matter what the etiology, the routine regimen of treatment leaves much to be desired regarding rapid reduction of pain, tenderness, inflammation, and edema. The use of phenylbutazone in our hands has been confined to patients with thrombophlebitis of the superficial veins of the extremities, and in addition, to patients who have had vena cava ligation for suppurative pelvic thrombophlebitis and have or develop a retrograde thrombophlebitis. In the latter when routine management by lumbar sympathetic blocks, antibiotics, and rest for the relief of pain, fever and edema had failed, phenylbutazone was used to afford complete relief of symptomatology.

Stein [4, 5] has suggested that this drug may be highly effective in the rapid control of the symptomatology of thrombophlebitis of the superficial veins of the extremities. In view of his encouraging reports, the use of phenylbutazone in the treatment of thrombophlebitis of the superficial veins of the extremities was initiated in November 1955, on the Tulane Unit, Division of Obstetrics and Gynecology at Charity Hospital in New Orleans.

CHEMISTRY AND PHARMACOLOGY

The search for an effective antipyretic with a minimum of side effects has been undertaken since the introduction of cinchona bark in 1640. Salicylic acid was discovered in 1875 to have antipyretic properties and has been studied extensively since that time as has the quinine molecule. Various synthetic compounds since have been introduced and studied both for clinical efficacy and toxicity. Phenylbutazone, which is a pyrazole compound, was introduced by Stenzl [9] in 1949. Originally phenylbutazone was used in combination with aminopyrine (irgapyrine or butapyrin) to promote the solubility of aminopyrine. Due to the toxicity of the combined drugs, phenylbutazone has been used alone since 1951.[6, 7] In a comparative toxicity study, Kidd [7] reported an incidence of toxic reactions in 26.7 per cent of cases using phenylbutazone alone as compared with 68 per cent using butapyrin.

Chemically phenylbutazone is 3,5 Dioxo-1, 2 di-phenyl-4-butyl pyrazolidine, a white or slightly yellow crystalline powder of bitter taste in the pure state which is insoluble in water but may be dissolved in alkalies, ethyl alcohol, and other organic solvents. The sodium salt is readily soluble in water. The route of administration may be either oral or parenteral depending upon the desired speed of reaction and gastrointestinal complications. Brodie [8] noted that in patients on a daily oral dose of 800 mgms. the plasma concentration from the fourth to the fourteenth day ranged from 60 to 150 mgm/liter but that the level in each individual subject was relatively constant and the plasma concentration failed to increase in proportion to an increase in daily oral dose after the dose exceeded 800 mgm/day. Even at a dose of 1600 mgm/day, the plasma level was only 10 per cent higher than at 800 mgm/day. The level at 800 mgm/day was slightly higher than at 400 mgm/day. Therefore the authors felt that there was little to be gained by increasing the dosage over 600 mgm/day. The dose for the intramuscular route is approximately 1 gram/day. By the intramuscular route, the rate of absorption is slower and peak plasma levels are not obtained until six to eight hours following injection. By the oral route, the plasma peak concentration is reached in about two hours and a plateau is reached in three to four days of oral administration which remains relatively constant thereafter. Following dis-

continuation of phenylbutazone the plasma concentration decreases at the rate of approximately 15 per cent per day.

Domenjoz and Wilhelmi as cited by Byron [9] and Currie [10] report the analgesic effects of phenylbutazone to be approximately equivalent to acetophenitidin; the antipyretic effects to be approximately equivalent to antipyrine; and the anti-inflammatory effect to be greater than that of cortisone or corticotropin.

The analgesic effects were studied in animal experiments by determining the threshold for stimulation of the dental pulp in rabbits. The anti-inflammatory effects were demonstrated by inhibition of edema produced by the injection of egg albumin or formalin into the legs of rats. Wilhelmi [11, 12] demonstrated anti-inflammatory effects of butazolidin by the delay in the appearance of erythema on the shaved backs of guinea pigs exposed to ultra violet light, and by the reduction in edema and capillary permeability in mice ears irritated with croton oil. Domenjos[13] also demonstrated a diminished capillary permeability to colloidal dyes and marked antihistaminic effects in the perfused rabbit ear and in the guinea pig using butazolidin. The antipyretic effects were shown using rats infected with *E. coli* organisms.

Steinbrocker [14] reports antiarthritic effects of a notable degree in 25 per cent of his cases of rheumatoid arthritis and that analgesia may usually be noted in one to three days with objective signs of improvement in three to seven days.

Phenylbutazone has been used as an antipyretic, analgesic and anti-inflammatory agent in a variety of clinical conditions: The various forms of arthritis, including rheumatoid arthritis, rheumatoid spondylitis, osteoarthritis, gout, and traumatic arthritis.[7-10, 14-22] In addition, it has been used for the symptomatology of Hodgkins disease,[23, 24] acute rheumatic fever,[25, 26] postoperative thoracotomy [27] and periarteritis nodosa.[18]

The route of metabolism and mode of action are known only to a limited extent. Phenylbutazone and cortisone are similar in action in that they are both anti-inflammatory, antipyretic, and produce the side reactions of edema formation with sodium retention and may reactivate peptic ulcers. Phenylbutazone, however, is a synthetic pyrazole derivative which will not maintain adrenal function in hypophysectomized animals; alter the 17-Keto-steroid urinary excretion; cause eosinopenia or lymphopenia; change the glomerular filtration rate; or change the insulin requirements of diabetics. Cortisone on the other hand is a steroid hormone which will maintain adrenal function and does not cause anemia, granulocytopenia, agranulocytosis or stomatitis.[8, 9, 15]

Phenylbutazone is rapidly and almost completely absorbed by the gastrointestinal tract [8] and the oral route has been used in this series for simplicity, rapidity of effect, and consistency of absorption. Kuzell [16] believes that the intramuscular administration gives a more rapid remission in cases of acute gout and Kidd [7] claims no gastrointestinal complications when phenylbutazone is given intramuscularly.

TOXICITY

The toxic reactions to phenylbutazone have varied considerably both in type and extent. The range in reported incidence of toxic manifestations is from 0 to 100 per cent depending upon the authors quoted. The most commonly reported toxic reactions were gastrointestinal disturbances, including peptic ulcer hemorrhage and perforation, fluid retention and edema; blood changes to include leukopenia, thrombocytopenia, anemia, and agranulocytosis; and dermatologic changes such as morbilliform rash and exfoliative dermatitis.[6-10, 14-24, 28-39] It must be noted here that the majority of these patients were being treated for arthritic conditions for relatively long periods of time and usually following previous treatment with such drugs as salicylates, gold, and steroids which may have added to their complications. Regarding the problem of gas-

trointestinal disturbances, Bellomo and Fusco [40] reported that phenylbutazone orally acts as an immediate stimulus to the gastric mucosa to produce an increase in the quantity of the gastric juice secreted with a concomitant increase in acidity. The drug has been recommended to be given with or immediately following meals. Bunim [15] in a review of 2301 cases stated that there was a reported incidence of toxic reactions in 32 per cent of all patients taking phenylbutazone in which 38 per cent of these were severe enough to require interruption or termination of treatment. Currie [10] reports an incidence of 4.7 per cent most of which were mild and, out of 425 cases treated, only 3 required that the drug be discontinued. This low incidence of toxicity was attributed to careful dosage and selection of management for patients. Kuzell,[17] in a series to show the effects of prolonged administration of phenylbutazone listed 100 cases being treated for rheumatoid disorders over periods from twelve to fifty-four months for a total of 84,870 patient days at the usual daily dose of 300 to 400 mgms, listed only 6 cases in whom the drug had to be discontinued permanently because of untoward effects.

Of the 12 deaths listed as due to the administration of this drug there were 5 due to agranulocytosis,[41-45] 3 with peptic ulcers complicated by hemorrhage [17-42] or perforation,[46] 1 aplastic anemia,[47] 1 hypersensitivity,[48] 1 hypersensitivity with rash,[49], and 1 "gas gangrene".[46] In these 12 cases the age varied from 44 to 75 years, the total dosage ranged from 4 gms. to 414 gms. over intervals from 8 to 1034 days. THERE WERE NO DEATHS IN PATIENTS RECEIVING THE DRUG FOR LESS THAN EIGHT DAYS AND NONE WAS KNOWN TO HAVE BEEN RECEIVING EXCESSIVE DOSES. ALL BUT ONE OF THESE CASES WERE BEING TREATED FOR RHEUMATOID ARTHRITIS.

As we have used phenylbutazone in the treatment of thrombophlebitis, the dosages have been of a relatively low range and over a short-time period.

METHOD AND DOSAGE

It has been our practice to administer this drug in the following dosage schedule: 200 mgms. three times a day with meals for three days, then 100 mgms. three times a day with meals with the general rule that the drug be discontinued twenty-four hours following the cessation of symptoms. In the vast majority of patients, five days of therapy was all that was necessary to obtain the desired results.

At the initiation of phenylbutazone therapy a complete blood count, hematocrit, urinalysis, and microscopic studies of the urine were performed. These were repeated every other day until the termination of therapy. All of the patients except one were hospitalized and under close supervision. Examinations for dermatologic changes and symptoms of toxicity were made twice daily. The toxic manifestation of edema formation was not observed in this series. Reduction in preexisting edema was used as a criterion for our evaluation of the clinical response to phenylbutazone in thrombophlebitis. This may be explained by the anti-inflammatory effect of the drug upon the edema produced by the pathologic process.

PREDISPOSING FACTORS

Predisposing factors in the cases of thrombophlebitis treated in this series are listed in Table 1. Postoperative, postpartal, and postabortal cases comprise the majority in this group. Included are 6 cases in whom vein ligations had previously been performed for deep phlebitis, and retrograde thrombosis had developed. The problem of thrombophlebitis developing in va-

TABLE 1
ETIOLOGY OF THROMBOPHLEBITIS
BUTAZOLIDIN SERIES

Predisposing Factors	Cases
Postoperative	15
Postpartal or postabortal	10
Following intravenous therapy	8
Pregnancy with varicosities	2
Trauma to calf	1
Total	36

ricosities aggravated by pregnancy is serious and the clinical response shown by 2 patients was gratifying. Both subsequently delivered normal infants without difficulty weeks later. Intravenous administration of medications, blood, or fluids is always a potential etiologic factor in phlebitis and often may be difficult to control. Antibiotics are of little value in such chemically induced phlebitic processes. Phenylbutazone has been most valuable in this type of case. Usually within hours satisfactory results are obtained. It has been our experience that, when using other forms of therapy, symptoms persist for days or weeks. Three patients developed subsequent recurrences of phlebitis at later dates and repetition of phenylbutazone was necessary and effective.

LOCATION

Table 2 depicts the location and extent

TABLE 2
LOCATION AND EXTENT OF THROMBOPHLEBITIS

	Right	Left	Bilateral
Calf	7	13	3
Thigh	8	12	3
Arm	7	2	0

of the venous involvement in both superficial and deep veins. The occurence of the majority of cases in the right arm is due to the fact that the right arm is more convenient to use for intravenous fluids during surgery.

TYPE

As previously stated, the majority of patients in this series were treated for thrombophlebitis of the superficial veins. (Table 3) Six of these cases had deep

TABLE 3
TYPE OF THROMBOPHLEBITIS

Type	Right	Left	Bilateral
Superficial			
Calf	5	11	1
Thigh	6	10	1
Deep *			
Calf	2	2	2
Thigh	2	2	2

* Retrograde thrombophlebitis following vein ligation.

phlebitis. They were due to retrograde thrombosis following vein ligations.

PREVIOUS THERAPY

The methods of therapy utilized prior to the initiation of phenylbutazone therapy varied. Lumbar sympathetic blocks, antibiotics, anticoagulants and vein ligations, alone or in combination were used. Twenty-one cases who had had other methods of therapy and had failed to respond or in whom the results were not definitive, were placed on phenylbutazone therapy. In 15 cases phenylbutazone was used as the primary therapy. (Table 4).

TABLE 4
PREVIOUS THERAPY

Method	Cases
Antibiotics	19
Lumbar sympathetic blocks	7
Vein ligations	
Bilateral femoral	1
Vena cava—ovarian	5
Anticoagulants	1
Sulfa drugs	1
No therapy	15

TOTAL DOSAGE

Twenty-four hours after a clinical response had been obtained the drug was discontinued. The average total dose required was 2.3 gms. given over a period averaging slightly less than five days. The shortest duration of treatment was forty-eight hours and the longest sixteen days. Only one patient had a total dose exceeding 3.3 gms. (Table 5)

TABLE 5
DURATION OF TREATMENT AND TOTAL DOSAGE

Duration	Total Dose	Cases
2 days	1.2 gms.	5
3 days	1.8 gms.	6
4 days	2.1 gms.	7
5 days	2.4 gms.	3
6 days	2.7 gms.	6
7 days	3.0 gms.	7
8 days	3.3 gms.	1
16 days	6.6 gms.	1

CLINICAL EVALUATIONS

Several criteria were used in the evaluation of clinical results. The analgesic, antipyretic, and anti-inflammatory actions of phenylbutazone in phlebitis are listed in Table 6. The most rapid response, and usually the first to be noted by the patient, was relief of pain. Reduction in systemic temperature, local heat, and conversion of Homan's sign from positive to negative, were relatively rapid. Resolution of the

TABLE 6
CLINICAL RESPONSE TO BUTAZOLIDIN

Type Response	0-48 hrs.	48-96 hrs.	Over 96 hrs.
Edema reduction	12	13	3
Homan's sign reverted from pos. to neg.	12	2	2
Local heat reduction	24	6	2
Relief of pain	32	3	1
Temperature reduction	18	8	2
Induration reduction	6	11	2
Local tenderness reduction	19	10	7

disease process as observed by reduction of edema, induration and tenderness required a longer period of time. In the majority of cases there was complete resolution by the fourth day. Sixty-three per cent of all favorable results were demonstrated within two days and by the end of five days 90 per cent were recorded.

TABLE 7
CLINICAL EVALUATION OF RESPONSE

Results	Major Improvement in	Cases
Excellent	Less than 48 hours	21
Good	Less than 72 hours	11
Fair	Less than 96 hours	2
Poor	Over 96 hours	2

TOXIC REACTIONS

There were no serious toxic reactions observed in this series. One patient with a past history of a peptic ulcer developed mild epigastric pain after ten days of treatment but there were no signs of gastrointestinal bleeding. The epigastric pain subsided upon termination of therapy. Three patients showed a relative reduction in leukocyte counts after being under phenylbutazone therapy. Of these 3, 2 had been under therapy six days and one seven days. Reductions in leukocyte counts in these cases were not marked (13,200 to 4150, 9000 to 5000, 12,000 to 7100). These are actually within a range compatible with the clinical response to an inflammatory process. There were no signs or symptoms of fluid retention, dermatologic changes or other toxic reactions.

SUMMARY AND CONCLUSIONS

1. The preliminary work leading to the therapeutic trial of phenylbutazone in the treatment of thrombophlebitis is discussed.

2. Thirty-six patients * were treated using moderate dosages over short periods. Our methods of therapy, observations for possible toxic reactions, and clinical results are presented.

3. Symptomatic response was excellent and there were no toxic manifestations of note.

4. The encouraging results shown in this series suggest further clinical trial of this drug in the hospital treatment of phlebitic processes and a final evaluation as to clinical efficacy in relation to toxicity formulated.

REFERENCES

1. Collins, C. G., MacCallum, E. A., Nelson, E. W., Weinstein, B. B., and Collins, J. H.: Surgery 30:298, 1951.
2. Collins, C. G., Ayres, W. B.: Surgery 30:319, 1951.
3. Collins, C. G., and others: Surgery 31:528, 1952.
4. Stein, I. D., and Rose, A. O.: Arch. Int. Med. 93:899, 1954.
5. Stein, I. D., and Rose, A. O.: Circulation 12:833, 1955.
6. Kuzell, W. C. and Schaffarzick, R. W.: Bull Rheum. Dis. 3:41, 1952.
7. Kidd, E. G., and Freyberg, R. H.: Ann. Rheum. Dis. 11:20, 1953.
8. Brodie, B. B., and others: Am. J. Med. 16:181, 1954.
9. Byron, C., Orenstein, H.: New York State J. M. 53: 676, 1953.
10. Currie, J. P., Peebles Brown, R. A., and Will, G.: Ann. Rheum. Dis. 12:88, 1953.
11. Wilhelmi, G. Schweiz: Med. Wchnschr. 79:577, 1949 (cited by (9)&(10).
12. Wilhelmi, G. Schweiz: Med. Wchnschr. 80:936, 1950 (cited by (9)&(10).
13. Domenjoz, R.: Gen. Pract. Clin. 105:467, 1952 (cited by (9)&(10).
14. Steinbrocker, O., and others: J.A.M.A. 150:1087, 1952.
15. Bunim, J. J., Sokoluff, L., Williams, R. R., and Black, R. L.: Ann. Chron. Dis. 1:178, 1955.
16. Kuzell, W. C., and others: Am. J. Med. 16:212, 1954.
17. Kuzell, W. C., Schaffarzick, R. W., Naugler, E. W., and Champlin, R. M.: New England J. Med. 256:388, 1957.
18. Kuzell, W. C. and Schaffarzick, R. W.: California Med. 77:319, 1952.
19. Kuzell, W. C. and Schaffarzick, R. W.: J.A.M.A. 149:729, 1952.
20. Stephens, C. A. and others: J.A.M.A. 150:1084, 1952.
21. Davies, H. R., Barter, R. W., Gee, A., Hirson, C.: Brit. M. J. 2:1392, 1952.
22. Granirer, L. W.: J.A.M.A. 150:1332, 1952.
23. Rottino, A., Joffe, A., Hoffman, G.: A.M.A. Arch. Int. Med. 93:561, 1954.
24. Bichel, J.: J.A.M.A. 161:1095, 1956.
25. Kalliomaki, L.: J.A.M.A. 161:103 (ab) 1956.
26. Fleming, J., and Will, G.: Ann. Rheum. Dis. 12:95, 1953.
27. Farinos, E. C., and Zozaya, G.: J.A.M.A. 161:1706 (ab) 1956.
28. Fishman, H. C., Reynolds, J.: J.A.M.A. 153:642, 1953.
29. Charet, R. and Siegle, I.: J.A.M.A. 151:556, 1953.
30. Stifel, J. and Burnheimer, J. C.: J.A.M.A. 151:555, 1953.

* Our thanks to Dr. Ambrose H. Storck for the use of one of his patients in this series.

31. Hinz, Carl: J.A.M.A. 151:38, 1953.
32. Bershof, E. and Oxman, A.: J.A.M.A. 151:557, 1953.
33. Werblow, S. C., and Neber J.: J.A.M.A. 151:1286, 1953.
34. Brandwein, S.: New York State J. Med. 53:1577, 1953.
35. Kranin, P.: J.A.M.A. 152:31, 1952.
36. Raffensperger, E. C.: J.A.M.A. 152:30, 1953.
37. Shields, W. E., Adamson, N. E., MacGregor, J. B.: J.A.M.A. 152:28, 1953.
38. Steinbrocker, O.: J.A.M.A. 151:143, 1953.
39. Nassim, J. R.: Brit. M. J., 1:1310, 1953.
40. "Foreign Letters"—J.A.M.A. 161:173, 1956.
41. Dilling, N. V.: Lancet 1:1230, 1953.
42. "Medico-Legal Notes" Brit. M. J. 2:1427, 1952.
43. Steinberg, C. I. Bobrod, M. G., and Roodenburg, A. L.: J.A.M.A. 152:33, 1953.
44. Etess, A. D., and Jacobson, A. S.: J.A.M.A. 151:639, 1953.
45. "Medicine and the Law", Lancet, 1:192, 1953.
46. Benstead, J. G.: Brit. M. J., 1:711, 1953.
47. Leonard, J. C.: Brit. M. J., 1:1311, 1953.
48. O'Brian, D. J., and Storey, G.: Brit. M. J., 1:792, 1954.
49. Nathan, D. A., Meitus, M. L., Capland, L., and Lev, M.: Ann. Int. Med. 39:1096, 1953.

DIAGNOSIS AND TREATMENT OF PARTHENIUM DERMATITIS *

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NEW ORLEANS

Contact dermatitis due to sensitization to members of the Rhus family (poison ivy and oak) has long been recognized. While this plant is known to be highly antigenic, it is not sufficiently emphasized that many other plants frequently produce sensitization. A common mistake is to classify individuals with plant dermatitis with the rather vague diagnosis of "poison ivy." The skin lesions in plant dermatitis are not ordinarily morphologically different unless the exposure is mainly in one particular area, such as a housewife who has dermatitis of the hands due to contact with some plant used in cooking. The fact that other plants may be etiologically incriminated explains immediately the poor therapeutic results obtained from hyposensitization in many cases of "poison ivy" when patch tests are not done.

Patch testing should not be attempted until the acute phase of the dermatitis has subsided. Until recently one had to rely mainly on ointments, lotions, etc. for symptomatic relief. The advent of the corticosteroid hormones has facilitated easier handling of the acute phase. Topical therapy or hormone therapy may be used together or separately.

The purpose of this presentation is to emphasize the need to patch test for possible sensitization to other plants, including *Parthenium hysterophorus*, as a common cause of dermatitis. This plant which resembles ragweed is ubiquitous in many parts of the country especially in the South. It is very common in Louisiana, and frequently grows in vacant lots and by the side of roads. It is also called "feverfew".

CASE REPORTS

C. B. S., male, forty-four years old, dairy employee, had a generalized dermatitis of long standing which improved when he left home or work, or was hospitalized in Charity Hospital. In spite of treatment the skin lesions would always reappear when he returned home. Patch testing revealed a very strong reaction to the parthenium leaf. Hyposensitization was begun with alcoholic extract of parthenium and he was instructed to avoid all contact with the plant. Improvement was satisfactory and he did not have any recurrence as long as he remained under our supervision in New Orleans.

Mrs. G. B., female, twenty-nine year old housewife, who lives in a suburban area of New Orleans,

* From the Department of Medicine, Louisiana State University, School of Medicine, New Orleans.

had a history of recurrent dermatitis for most of her life. Routine dermatologic treatment gave temporary relief. Her lesions were on exposed areas such as hands, arms, legs, and feet. Respiratory allergy was also present. A positive patch test was obtained with parthenium and hyposensitization was then commenced. In addition, she was also treated with extracts of house dust, giant and short ragweed and true marsh elder. Excellent therapeutic results were obtained with this routine.

A. L., male, fifty-two year old truck driver from rural Louisiana, had dermatitis mainly on exposed areas, although lesions were present on the thighs. Previous treatment gave only temporary relief. This patient showed a positive parthenium patch test. Hyposensitization with parthenium extract has relieved symptoms to a great degree, and no additional medication is now needed.

R. L. D. male, thirty-four year old truck driver from rural Louisiana, had a history of dermatitis of seven years' duration. The dermatitis at times appeared all over the body, but was mainly localized on the hands. Routine dermatological treatment gave little help, necessitating prednisone for several months. He gave a positive reaction to parthenium extract. There was also a history suggestive of bronchial asthma. He gave a positive skin test reaction to house dust, and was treated with this extract in addition to alcoholic extract of parthenium. He has improved during hyposensitization; however, minor lesions have continued on the hands, which made it necessary to continue prednisone. It must be pointed out that it is impossible for him to completely avoid contacts with this plant during his daily work.

It is proper to point out that contact with an offending plant may not be recognized by the patient. Exposure may be through some unsuspected object, such as a dog which has been in contact with the offending plant, wet clothes on an outdoor line may be impregnated with particles of the plant which have been carried by the wind, etc. Contacts of this type are difficult to trace or appreciate.

The following case histories illustrate prompt symptomatic relief which was obtained in cases of acute allergic dermatitis.

Mrs. R. D., female, forty-nine year old clerical worker. This patient had a history of acute dermatitis for one week prior to her first visit. Lesions were mainly on the wrists, neck, ankles and legs. She suspected sensitization to poison ivy. Forty units of cortrophin-zinc were given to her on her first visit and repeated the following day. By the third day the lesions had greatly improved. It will now be necessary to make a specific diagnosis, (by patch testing) to be probably followed by hyposensitization.

Mrs. E. A., a fifty-six year old female clerical worker, had recurrent lesions the distribution of which resembled atopic dermatitis. Urticarial wheals were also present at times. She had not been relieved by various medications including PBZ, intravenous calcium gluconate, prednisone, etc. Forty units of cortrophin-zinc were given and repeated in forty-eight hours and marked improvement occurred after the second injection. Three more injections of cortrophin-zinc were given with continued improvement. Allergy work-up is yet to be completed on this patient.

Cortrophin-zinc, which contains 40 units of ACTH per cc., has proved to be an effective agent for symptomatic relief. It is easy to administer, may be given with a small gauge needle, and its effects are relatively long-lasting.

SUMMARY

1. So called "poison ivy" dermatitis may be due to allergy to other plants.
2. *Parthenium hysterophorus* sensitization has been found to be a common cause of contact dermatitis of the plant type.
3. ACTH has been found to be of help in obtaining rapid symptomatic relief in allergic dermatitis.
4. In plant dermatitis the specific etiological factor involved must be isolated at a later date by means of patch testing.

USE OF THE ATARACTICS IN MENTAL ILLNESS *

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MANDEVILLE

INTRODUCTION

At a psychopharmacology conference held last year in Washington a favorite corridor anecdote dealt with some advice said to have been given by Nolan D. C. Lewis, a very well known psychiatrist, to a group of younger colleagues and students. When asked for his opinion of the newer psychiatric drugs, Dr. Lewis is reported to have said, "Better use 'em quickly, boys, while they still work."

* Presented at the Seventy-seventh Annual Meeting of the Louisiana State Medical Society, May 8, 1957, New Orleans.

The cycle of enthusiasm, followed by skepticism, followed by sober evaluation through which a new treatment method goes is widely recognized. The present writer and a number of his co-workers have admittedly entered the second phase, that of doubt and suspicion. The views which follow, then, represent current opinion and are subject to revision and correction in the final phase of the cycle.

CLINICAL IMPRESSIONS

The first portion of this report contains: clinical impressions regarding a number of drugs of special interest to psychiatry, personal impressions of the writer and of other members of the psychiatric staffs of the Louisiana state hospitals. Informal conversations during the course of work as coordinator of a drug research program, still underway, have yielded many opinions and, in addition, responses to a formal questionnaire have been used.

One clinical impression is particularly outstanding. Every physician responding to our questionnaire uses chlorpromazine (Thorazine) more frequently than any other psychopharmacologic agent. Most report that their use of this compound has increased markedly over the past year, often with a corresponding decrease in the use of other drugs. Figure 1 shows

FIGURE 1

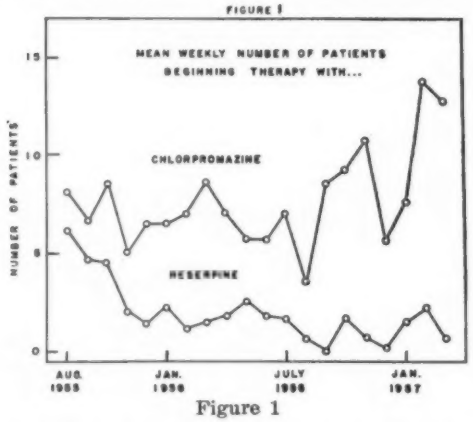

Figure 1

this trend at the Southeast Louisiana Hospital. It will be seen that during late 1955 chlorpromazine and reserpine were competing for popularity. During late 1956 and early 1957, however, it is clear that chlorpromazine is far and away the outstanding choice. The preference for chlorpromazine is sufficiently great on the part of certain physicians that they report the conviction that it is the only psychopharmacologic agent which, in their experience, exerts any significant desirable effects other than those attributable to suggestion.

As implied above, opinions about most of the other compounds are considerably more varied. The use of reserpine (Serpasil, Sandril) has definitely diminished. For a time there was considerable opinion that reserpine was equally as effective as chlorpromazine but merely slower and less dramatic in its action. In recent months this idea has been expressed less often and one hears increasing doubt regarding the real value of this compound for modifying psychiatric symptoms. Meprobamate (Equanil, Miltown) has been rather extensively used. It has, of course, been recommended chiefly for the milder, less severely disturbed patient with moderate anxiety and tension. Impressions would certainly agree that it is ineffective in more severe agitation. Impressions regarding its usefulness even in those patients for which it is specifically recommended vary, however. It must be said that there is increasing doubt as to its effectiveness. Promazine (Sparine), a relatively recent introduction, is currently enjoying some popularity as a substitute for chlorpromazine in cases where that drug proves toxic or ineffective. It is generally agreed, however, that promazine is a second choice to chlorpromazine and doubts about its efficacy are frequently heard. Experience with a number of the still newer ataractics is much too limited to justify individual reports. I refer to such compounds as mepazine (Pacatal), ectylurea (Nostyn), hydroxyzine (Atarax), phenaglycodal (Ultran), and proclorperazine (Compazine). However it may be said that no enthusiasm for any of these compounds, with the possible exception of proclorperazine, has been ex-

pressed by the physicians whose impressions are included in this study.

We may take a moment to discuss several compounds which are not, strictly speaking ataractics: azacylonal (Frenquel), pipradol (Meratran) and methylphenidylacetate (Ritalin). Of these Ritalin is being rather extensively used, particularly at the Central Louisiana State Hospital and particularly in combination with chlorpromazine, for the relief of depression. Others report considerably less success with this drug. On the basis of very limited experience, the remaining compounds in this group have not aroused any noticeable enthusiasm. At the Southeast Louisiana Hospital, under the direction of Dr. Frank Ervin, we have currently under investigation a new, experimental drug not yet released by the McNeil Laboratories of Philadelphia. This compound, in a very few cases, has seemed to be sufficiently effective in relieving depression to warrant its further study.

With regard to the dosage levels in common use we find a distinct trend toward the use of larger doses. This is particularly true in the administration of chlorpromazine which is now commonly prescribed in doses up to 2400 mg. per day. Many of the physicians reporting indicate that they have had numerous cases which failed to respond to lower doses of this drug but which showed improvement after the dosage level was increased. It is felt that reluctance on the part of physicians in the community to use these larger doses may occasionally result in the unnecessary commitment of patients who could be successfully treated at home. As with any drug, adequate dosage is of the greatest importance.

The matter of reluctance to use higher doses, particularly of chlorpromazine, leads immediately to a consideration of the toxicity of this drug. First of all it may be said without hesitation that chlorpromazine is the most toxic of the compounds under consideration. Nevertheless, we have had no fatalities with this drug and, at the Southeast Louisiana Hospital, no cases of jaundice or agranulocytosis, its two most dreaded complications. The other hospitals have reported a number of chlorpromazine-treated cases which developed reversible clinical jaundice. Another side-effect of chlorpromazine therapy is a Parkinsonian-like syndrome which can at times be quite troublesome to the patient but usually responds to treatment with Artane and does not usually necessitate termination of therapy. Similarly, urticarial rashes, often combined with extreme photosensitivity, have frequently been observed in our chlorpromazine-treated cases but usually respond to topical medication and some modification of activity. Only rarely has it been necessary to discontinue therapy because of skin eruptions. Hypotensive reactions with falling and fainting occur occasionally during the initial days of chlorpromazine therapy and are adequately managed by a few days of bed rest. Nasal congestion, dryness of the mouth, diarrhea and constipation are occasionally seen but respond well to general measures and are not considered of special importance. There is one side-effect of chlorpromazine therapy which requires particular mention. This is a reaction consisting of increased agitaton and apprehension on the part of the patient, usually experienced within the early weeks of therapy and often described by the patient as a jittery feeling or just 'being scared'. Mild to moderate exacerbations of depressive tendencies sometimes occur. Occasionally tremors are seen. The patient usually attributes these symptoms to the medication and frequently demands its discontinuation. Depending upon individual considerations we have discontinued the drug or attempted to continue medication on through this period, which is usually transitory.

Reserpine is the only other compound in this group with significant toxicity and it is considerably less toxic than chlorpromazine. The two drugs share the tendency to produce hypotensive reactions, Parkinsonian-like syndromes and mild depression. Both drugs also often produce peri-

ods of drowsiness and lethargy but these are usually transitory and do not usually require discontinuation of therapy.

We have felt that the toxic reactions observed by us and reported in the literature, while in no sense a deterrant to the employment of these drugs in adequate doses, do necessitate routine, periodic laboratory examinations of the blood, for hematopoietic inhibition, and the urine, for retention of bile and bilirubin.

With regard to the specific areas of efficacy of the ataractic drugs, and I shall speak particularly of chlorpromazine, it is generally agreed that, when effective, they do have a calmative, tranquilizing effect on patients with distinct psychomotor hyperactivity and anxiety. In addition they often seem to enable the disorganized, incoherent patient, with or without delusional ideation and hallucinations, to reintegrate his mental processes. There is some opinion that this latter effect may be secondary to the lessening of anxiety and hyperactivity and the fact that the patient becomes thereby more accessible to stimuli from his environment and contact with his therapists. There is some evidence, however, that the facilitation of reintegration occurs even in withdrawn and emotionally flattened patients who do not show overt anxiety as a part of their presenting picture.

It is our opinion that the drugs do not cure schizophrenia or any other mental illness but sometimes relieve certain of the distressing symptoms which might otherwise cause the patient to be involved in a descending spiral of disorganization and eventual deterioration. By relieving these symptoms they render the patient more accessible to his environment and to other forms of therapy, and permit the utilization of his own natural recuperative powers. The importance of emotional support for the eventual adjustment of a patient 'coming out' of a psychotic state, whether this remission be aided by drugs or not, cannot be overemphasized. Without question the use of these drugs reduces greatly the need for other symptomatic therapies such as ECT and insulin. In a great many instances the "social milieu" of the ward is improved.

Opinions differ as to the eventual place of the psychopharmacologic agents. At present we believe that the ataractic drugs will probably find a definite *but distinctly limited* place in hospital psychiatry. We predict that they will eventually be assigned their proper niche as one of many types of symptomatic therapy.

A STUDY OF CHANGES IN PSYCHIATRIC STATUS

I should like now to turn briefly to a very simple and in many ways inadequate study which was done during the period from August 1955 to June 1956, in the Louisiana State Hospital System. All patients receiving reserpine or chlorpromazine during this period received an initial, pretreatment rating of psychiatric impairment on a five point scale (none, minimal, mild, moderate, severe) and a final psychiatric rating at the time of exit from the hospital or at the end of the period of study.

There were a number of grave defects in this study. No provision could be made for control. Only a single pretreatment and a single post-treatment rating were obtained so that there was no possibility of determining an interrater reliability. The ratings were made by different observers with no background of agreement on the definitions of the various terms. Courses of therapy varied widely. There was neither a single fixed-dose or fixed-length course nor were there uniform criteria for the regulation of doses and lengths of treatment based on individual patient-responses to the drugs. For these reasons, and because of a number of specific difficulties which arose in carrying out the research, the results to be reported must be considered of a very preliminary and tentative nature. This study is now being repeated with an effort to eliminate many of the defects inherent in the previous research. Nevertheless we feel that the results are of sufficient interest that they may be briefly reported here.

Figure 2 summarizes the results of the

early study. Results with chlorpromazine and reserpine are reported separately. The striking finding shown in this graph is the fact that a very large percentage of the patients (44.9 for chlorpromazine and 70.5 for reserpine) showed no improvement whatever and that, among those patients showing improvement, by far the majority showed only one-step improvement, i.e. from severe to moderate, moderate to mild, mild to minimal, or minimal to none. Inspection of the graph shows that the number of patients showing "two-step" or greater improvement is very small. Stated in another way, these results show that only an infinitesimally small number of patients receiving the drugs showed improvement to the minimal or none levels of psychiatric impairment indicative of a complete remission or recovery. We feel that insofar as these results are valid and subject to our ability to repeat the experiment under more rigid conditions, this study supports our clinical impression that the ataractic compounds are not miracle drugs and do not, in themselves, even produce complete remissions in most cases but rather relieve a small portion of the patient's symptoms.

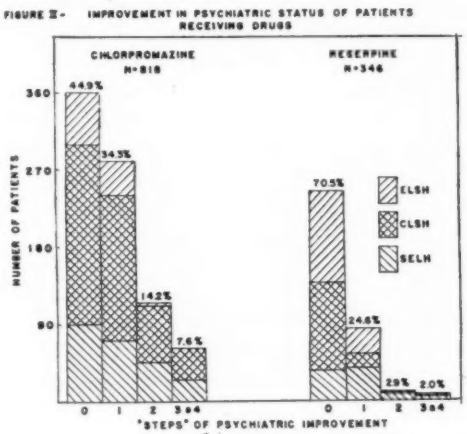

Figure 2

This point is emphasized not to disparage the value of symptomatic relief in medical therapy but to discourage the overconfident use of these compounds with the consequent neglect of other forms of psychiatric therapy now available and the neglect of research along other lines.

The Journal of the Louisiana State Medical Society

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PREGNANCY, GLYCOSURIA, AND DIABETES

Glycosuria is never trivial, and when it is found in pregnancy the need for proper explanation and appropriate handling is pressing.

The identification of its cause is not simple for it is not due to lactose, which appears only during lactation. Glycosuria developing for the first time in a woman who has become pregnant is probably not benign. Glycosuria in a pregnant woman is more difficult of interpretation than in the nonpregnant individual because pregnancy lowers the renal threshold for sugar and allows the appearance of sugar in the urine under a variety of conditions. Among these are some which the pregnancy itself may cause to become urgent.

The concept of a renal threshold for the excretion of sugar in the urine is a convenient basis for considering the various forms of glycosuria. It is the concentration of sugar in the blood which must be reached for abnormal amounts of sugar to appear in the urine, and is generally considered to be 140 to 160 mg. per 100 ml. whole blood and 170 mg. per 100 ml. for plasma. It is thought that a rigid barrier does not exist but that the "threshold" is the end result of glomerular filtration, on the one hand, and tubular reabsorption on the other. This value will be influenced by many factors—one of which is pregnancy. It has been considered that the pituitary influence is responsible for lowering the threshold in pregnancy.

The least threatening of these conditions in pregnancy is renal glycosuria, which may be found in 10 to 15 per cent of normal pregnant women as the result of lowering of the renal threshold for sugar and allowing glucose to escape in the urine. The renal threshold for glucose returns to normal after the lactation period is ended. Renal glycosuria is not a forerunner of diabetes. Its presence is identified by the determination of two blood sugars an hour apart, and the determination of the urine sugar midway between. If the blood sugars are normal and the urine shows significant sugar, renal glycosuria is demonstrated. The blood sugars of a glucose tolerance test are normal in the nondiabetic patient having renal glycosuria.

The lowering of the threshold for sugar in the pregnant state is of importance in other respects. One of these is that pregnancy may raise subclinical signs of prediabetes to the clinical level. Following the discovery of glycosuria in a pregnant woman, dependence should not be placed on the determination of a single blood sugar. During pregnancy the fasting blood sugar level is 10 to 20 mg. lower than it is in the nonpregnant woman. A single

blood sugar determination, therefore, may be misinterpreted as renal glycosuria of pregnancy. In such a situation a glucose tolerance determination is necessary, and this should be repeated in each trimester of the pregnancy.

Large sized infants are common in prediabetic pregnancy, which may also be complicated by high incidence of stillbirths, premature deliveries, hydramnios, and toxemias. These abnormalities may occur many years before clinical diabetes is manifest. The mother of a newborn weighing over ten pounds should have glucose tolerance test. The prediabetic glucose tolerance abnormality frequently disappears within forty-eight hours of delivery. Moss [1] states that in the postpartum period one-third of these mothers, however, will still have diabetic glucose tolerance curves, but if they are tested during the middle third of a subsequent pregnancy the majority will have decreased carbohydrate tolerance. He also states that if the husbands of the nondiabetic mothers of large babies are tested approximately one sixth will have diabetes.

Another situation in which the lowering of the renal threshold for sugar in pregnancy is of great clinical importance is the production of renal glycosuria in the established diabetic. In this condition, renal glycosuria causes serious difficulty in the management of the diabetes during pregnancy. Perkoff and Tyler [2] have reported observations on ten diabetic patients, among whom five exhibited renal glycosuria. This led to severe hypoglycemic episodes, and subsequently contributed to the occurrence of ketosis in three of these patients.

There are various theories about what may contribute to the well known readiness with which pregnant diabetics become ketotic. Among these, it is reasonable to expect that when the pregnant diabetic develops renal glycosuria, rapid loss of glucose in the urine would be conducive to ketosis and acidosis.

Before the advent of the insulin era, pregnancy in the diabetic patient was not a clinical problem for many months because of abortions. Now, among the hundred thousand women of child bearing age in this country it is expected that about ten thousand will become pregnant. Among these thousands and among the vast number of pregnant women who will show glycosuria, the clinical problem of identification of its cause is of pressing importance. In each of these its clarification will become possible through the use of the glucose tolerance test. The criteria for the recognition of diabetes or renal glycosuria, or both, are the same in the pregnant and in the nonpregnant patient.

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[1] Moss, James M.: Diagnosis of diabetes mellitus, J. Student A.M.A. 4:19 (February) 1955.

[2] Perkoff, G. T., and Tyler F. H.: Renal glycosuria in pregnant diabetic patients, Arch. Int. Med. 97: 758, 1956.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

SCIENTIFIC PROGRAM
1958 ANNUAL MEETING

Listed below are chairmen of the various scientific sections for the 1958 Annual Meeting which will be held in Shreveport, May 5-7. Members who are desirous of presenting papers at this meeting should promptly contact the chairman of the respective section before which they wish to appear since plans for the program are being formulated at this time, and preference is given in order of requests received.

Allergy—Dr. Henry D. Ogden, New Orleans
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Surgery—Dr. Paul D. Abramson, Shreveport
Urology—Dr. John G. Menville, New Orleans

IMPORTANT

Both volumes of the Rudolph Matas History of Medicine in Louisiana have been available for several months. The Committee had 2000 copies of Volumes I and II printed.

Up to the present time only 188 copies of Volume I and 173 copies of Volume II, have been purchased by members of the State Society. The number of volumes printed was based on the approximate membership of the State Society, and the cost for each volume is $5.00 plus tax. Remittance should be sent to J. A. Majors Company, 1301 Tulane Avenue.

The Committee was hopeful that the response would be much greater than it has been.

The entire expense for editing and publishing of the Rudolph Matas History of the Louisiana State Medical Society was borne by a Trust Fund which was created by Dr. Matas.

None of the volumes have been offered for sale outside of the profession up to the present time. If the profession does not avail itself of the opportunity it will be necessary for the Trust Fund Committee to offer these books for sale to the public.

It is our hope that the members of our Society will forward their orders for these volumes without delay, as we feel every member of our organization should have a copy of these volumes.

Dr. Isidore Cohn, Chairman
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MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Indepencence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

GASTROENTEROLOGICAL CONVENTION

The 22nd Annual Convention of the American College of Gastroenterology will be held at The Somerset in Boston, Mass., on October 21, 22, 23.

In addition to the many individual papers to be presented, there will be panel discussions on Chronic Ulcerative Colitis, Diseases of the Esophagus, Peptic Ulcer and the Management of Massive Gastrointestinal Hemorrhage in Patients with Liver Diseases. There will again be scientific as well as commercial exhibits and the sessions will be open to all physicians without charge.

On October 24, 25 and 26, immediately following the Convention, Dr. Owen H. Wangensteen of Minneapolis, Minn. and Dr. I. Snapper of Brooklyn, N. Y., will again be the moderators of the Annual Course in Postgraduate Gastroenterology. The sessions will be held at The Somerset and in the Joslin Auditorium of the New England Deaconess Hospital. Attendance at the Course will be limited to those who have registered in advance.

This year marks the Twenty-Fifth Anniversary Year of the College and silver certificates are to be presented to those who have been affiliated with the organization since its inception.

Honorary Fellowships are to be presented to Dr. Chester S. Keefer, Boston, Mass., Dr. William W. Frye, New Orleans, La., Dr. Stafford L. Warren and Dr. Rafe C. Chaffin, both of Los Angeles, Calif.

Copies of the program and further information concerning the Postgraduate Course may be obtained by writing to: American College of Gastroenterology, 33 West 60th St., New York 23, N. Y.

FORESEE RELIEF FOR REACTIONS TO PENICILLIN

Relief from adverse penicillin reactions, estimated to affect a minimum of 600,000 persons annually, may be available before the end of this year.

Two Midwest physicians, in papers presented at the American Medical Association Convention, reported highly successful results in the treatment of penicillin reactions by the injection of a newly developed penicillinase. Capt. G. M. Davis, Medical Corps, U. S. Navy, of Great Lakes Naval Training Station, and Dr. R. M. Becker of Madison, Wis. each reported that, in a significant number of clinical tests, allergic manifestations of penicillin reactions promptly receded following the injection of penicillinase.

Dramatic relief occurred within a period ranging from a few hours up to 24 hours and most patients had complete freedom from itching and swelling in 24 to 72 hours, they reported. An additional advantage of the new therapy is the absence of recurrence of symptoms even when other supportive therapy is stopped.

For these reasons, Dr. Becker told his A.M.A. audience, "penicillinase should be kept on hand

in every doctor's office or hospital where penicillin is administered."

ASPIRIN LESSENS REBOUND EFFECTS OF CORTISONE

Patients with rheumatic fever should be given aspirin to reduce rebound effects that occur when cortisone or related steroid hormones must be discontinued.

Dr. Edward E. Fischel, director of medicine at The Bronx Hospital, New York City, recently gave results of a study of combined use of aspirin and the hormones, cortisone, hydrocortisone and prednisone. The report was made at the 9th International Congress on Rheumatic Diseases, June 23-28, attended by more than 1,000 rheumatologists from 44 countries.

"When hormone therapy was stopped and aspirin therapy continued, no increase occurred in the severity of the rheumatic symptoms," Dr. Fischel's paper noted. "We were then able to continue the aspirin for a long period of time in an attempt to keep rheumatic activity suppressed."

Combined use of aspirin with cortisone, or the other steroids, was necessary because the rebound that often happens following withdrawal of these hormones "is not always mild," Dr. Fischel said.

POLIO VACCINE FOR HERPES SIMPLEX

Poliomyelitis vaccination may be of some value in the future treatment of recurrent herpes simplex, report Drs. Leon Adoni and Donald N. Tschan, Department of Dermatology, Temple University Medical Center. One man, 25, with a history of repeated attacks of herpes simplex since childhood, has had no recurrence of his fever blisters since the accidental ingestion and swallowing of liquid cultures of the three types of polio virus. Another man, 20, has had no recurrence of fever blisters since he was given two standard injections of vaccine during the summer of 1956. — Correspondence, **J.A.M.A.**, May 11, 1957.

YOUNG ADULTS AS WELL AS OLD NEED PROCTOSIGMOIDOSCOPIES

Proctosigmoidoscopy should be included as part of the routine physical examination of young adults as well as those above 35 or 40, according to three U. S. Air Force physicians who examined 500 subjects at the 3700th USAF Hospital, San Antonio, Tex. The 500 males ranged in age from 17 to 25. They were completely asymptomatic.

The investigators discovered that eight subjects had rectal polyps, and two had multiple polyps. Although all the polyps were benign, the physicians agree with others that "the benign adenomatous polyp of the rectum and rectosigmoid is a precancerous lesion." They "urge strongly that proctosigmoidoscopy be included in the complete physical examination of all patients."

In preparing for the 500 sigmoidoscopies, two kinds of enemas were given. Isotonic saline enemas were used with 112 subjects, but since "usually two enemas were required," the process was found to be "time-consuming, often technically difficult," and frequently there was a failure "due to lack of cooperation." The physicians then adopted the Fleet Enema Disposable Unit.

Administered to 388 patients 10 to 60 minutes prior to examination, the Fleet Enema (a 135 cc. packaged enema containing in each 100 cc., 16 gm. sodium biphosphate and 6 gm. sodium phosphate) "resulted almost always in a clean field making adequate examination quick as well as easy."

(Abbot, F. K., Monroe, L. S., and Spencer, F. M.: The incidence of polyps in 500 proctosigmoidoscopies in asymptomatic young men. Gastroenterology 32:704 (Apr.) 1957.)

RESEARCHERS NOTE EXCEPTION TO BIOLOGICAL LAW

A biological law which says that the living body always refuses to produce antibodies against itself—although it does produce them against substances from outside—may have an exception.

Evidence that chronic inflammation of the thyroid gland is caused by this exception to the rule was reported in the July 27 Journal of the American Medical Association by Dr. Ernest Witebsky and associates of the University of Buffalo School of Medicine and Buffalo General Hospital.

Their findings and method of study might also reveal the mechanism behind other diseases suspected, but not proved, to occur in the same way, they said. These include certain diseases of the blood known as dyscrasias (such as hemolytic anemia, in which red blood cells are destroyed).

The researchers explained that for many years only "foreign" substances from other species were considered as real producers of antibodies (materials in the blood which act against other substances, as polio antibodies act against polio virus). For instance, horse serum stimulates antibody production when injected into humans, but not horses. This is the basis for using animal serum against human diseases like smallpox.

Although research has indicated it is possible for antibodies to appear which are directed against members of the same species, there has

been no proof that this might occur within the same individual.

Now, however, Dr. Witebsky and associates have reported evidence that extract from the thyroid gland can produce this antibody effect "within the same species and even within the same individual under certain experimental conditions."

They succeeded in producing autoantibodies antibodies against the self) in dogs, rabbits, and guinea pigs by injecting them with their own thyroid extract. The material which apparently caused the reaction was thyroglobulin, a material known to contain antithyroid substances. They also discovered that injections produced considerable damage to the cell structure of the animals' thyroid glands.

POSTMORTEM CHANGES CONFUSE BLOOD ALCOHOL TESTS

Because alcohol may diffuse out of the stomach after death, special care must be taken in measuring alcohol levels in the blood during autopsy. Blood alcohol levels are commonly determined by taking blood from the sac surrounding the heart during autopsy, Drs. Henry W. Turkel and Houghton Gifford said in the July 6 Journal of the American Medical Association.

However, they have discovered that the alcohol level of blood in this pericardial sac is frequently higher than the level in the femoral vessels of the abdomen and legs. This happens because alcohol ingested just before death diffuses out of the stomach into the nearby areas after death, they said. The postmortem diffusion process does not extend as far as the femoral vessels.

Knowledge of the blood alcohol level at the time of death is frequently very important in criminal or civil court proceedings. Because of the "gravity of decisions" attached to these determinations, they must be accurate, the authors said.

Since determinations made from blood drawn from the heart area may be inaccurate, they recommended that blood from the femoral vessels be used instead.

The authors noted that these errors in measuring blood alcohol levels do not occur in examining the blood of the living, since tissue barriers are still intact and the circulating blood provides for a uniform distribution of alcohol throughout the blood stream.

Dr. Turkel is from the coroner's office of the City and County of San Francisco and Dr. Gifford is from the department of pathology, Stanford University School of Medicine.

A.M.A. COUNCIL ISSUES MEPROBAMATE WARNING

The American Medical Association's Council on Drugs today issued a warning about the potential hazards of the tranquilizing drug meprobamate (Miltown, Equanil).

The report in the July 20 Journal of the American Medical Association said the drug's extensive use since its introduction two years ago has been "based on the assumption that large doses of the drug can be administered with practically no side-effects." However, as the use has multiplied, it has become increasingly apparent that meprobamate is capable of producing "a rather wide variety of side-effects and untoward reactions."

The listing of adverse side-effects does not necessarily mean that the "usefulness of meprobamate is outweighed by its potential side-effects," the report said. It is intended to point out that side-effects and untoward reactions "can and do occur and that the drug should be administered with the same discretion" as other drugs.

Hypersensitivity reactions, including skin rashes, itching, shaking chills, and fever, have occurred with sufficient frequency to indicate that these are definite and relatively frequent complications, the report said.

There have been several reports of acute meprobamate intoxication, resulting usually from deliberate swallowing of very large amounts of the drug. None of these attempts at suicide have been successful, but alarming central nervous system symptoms have occurred.

No definite antidote or treatment for overdosage has been devised; therefore it is important to prescribe meprobamate with discretion and in small quantities, if at all, for patients who may have suicidal tendencies, the report said.

There is also evidence that meprobamate possesses habit-forming properties. Withdrawal symptoms, including convulsions, have been observed when the drug has been discontinued after long use. In addition to this physical dependence, psychological dependence with a tendency toward excessive self-medication is "undoubtedly created" in certain patients, the report said. Some patients may also need larger and larger doses to maintain the same tranquilizing effect; the chances of overdosage and acute intoxication are then appreciably increased.

Since the drug is intended primarily for those with emotional instability, the possibility of emotional complications must be recognized for intelligent use of the drug, the report said.

A variety of other side-effects, including

drowsiness, stomach and intestinal upsets, and muscular reactions, also have been noted.

VOLUNTARY HEALTH INSURANCE

The number of people in Louisiana who are covered by voluntary health insurance reached a new high by July 1, the Health Insurance Council reported. The Council estimates that about 1,250,000 persons were protected by some form of insurance designed to help pay hospital and doctor bills.

This figure, the Council said, is part of the continued growth of health insurance throughout the country, which was revealed last May in its 11th annual survey of the extent of voluntary health insurance coverage for 1956. The number of people covered by some form of health insurance in the nation today is more than 118 million, or over 70% of the U. S. civilian population.

In releasing the findings of its survey, which is based on reports of insurance programs of insurance companies, Blue Cross-Blue Shield and other health care plans, the Council went on to say that there were 1,195,000 persons covered by hospital expense insurance in Louisiana as of December 31, 1956. The total for 1955 of the number of persons covered for expenses incurred while in the hospital was 1,122,000.

Surgical expense insurance, which helps to defray the cost of physicians' charges for operations rose to 1,055,000, as compared with 929,000 the year before.

Persons protected by regular medical expense insurance, providing for doctor visits for nonsurgical care, numbered 631,000 in 1956, as against the previous year's figure of 547,000.

The Health Insurance Council, which is a federation of eight insurance associations representing over 90% of the accident and health insurance business handled by insurance companies, stated that this growth reflects the desire of the people of Louisiana to help protect themselves against the cost of accident and illness.

WHAT GOOD IS THE ILO?

The May 18 issue of the A.M.A. Journal (Pages 325-328) carries a very interesting and informative article on the International Labor Organization. It was written by William L. McGrath, Cincinnati manufacturer who until recently served as the United States employer representative to the International Labor Organization governing body. The ILO membership consists of government, employer, and employee representatives.

In the Journal article, Mr. McGrath presents a strong case for the United States to withdraw entirely from the ILO.

Referring to the November, 1956, governing body meeting, Mr. McGrath said:

"The labor-government-socialist majority, now well in control, openly, triumphantly, and even jeeringly displayed their satisfaction in riding rough-shod over the free employers and relegating free enterprise to a secondary position in the ILO."

In his "conclusions" in the Journal article, Mr. McGrath said further:

"The tripartite structure of the ILO has long since become a myth. Besides government in the government group, we now have government in the employers' group and government in the workers' group. The ILO is now to all intents and purposes an international government organization run mainly along political lines under the domination of advocates of collectivism, with free employers possessing no influence of any consequence and serving chiefly as whipping boys for the communists and the socialists. . . .

"I believe we can accomplish far more outside the organization than we can within it. As long as we stay in the ILO, we must carry its label. Do we not participate in its meetings and its findings? How can we condemn something of which we are a part? But if we withdraw from the ILO, we could label it for what it is. We could explain to the world that we are opposed to the majority philosophy of the ILO, that we do not believe in it, that we stand for free competitive enterprise instead of socialism and communism."

The Journal article and Mr. McGrath's opinions certainly confirm the good judgment of the A.M.A. House of Delegates.

HAZARD OF JET AIRCRAFT MAINTENANCE LISTED

Some of the health problems encountered in repairing and maintaining jet planes, and some preventive measures, were discussed in the June 1 Journal of the A.M.A. by two Air Force physicians.

The most hazardous of these operations is the cleaning and repair of the aircraft fuel cells. In fact, it is potentially more dangerous than cleaning bulk gasoline storage tanks, according to 1st Lieut. Americo R. Lombardi (MSC), and Capt. Arthur S. Lurie (MC).

The fuel cells of the B-47 jet bomber are rubber-lined cubicles that fill cavities within the aircraft fuselage. They are difficult to reach and allow a very small amount of working room. Since most of the cells are connected, it is often necessary to crawl from one cell into another, thus penetrating deeper into the fuselage and away from fresh air.

These "extremely poor" working conditions present a number of hazards, including fire and explosion, acute intoxication from fuel vapors, systemic poisoning from tetraethyl lead (a fuel ingredient), skin reactions from direct contact with petroleum hydrocarbons, and the acute psychological problem of confinement in a small space.

Seven of 12 airmen studied at Smoky Hill Air Force Base, Salina, Kan., reported various physical symptoms when they did not wear a protective face mask. These included dizziness, indigestion, headache, visual blurriness, "echoing," and repetition of thoughts.

Because these health problems may in the "not-too-distant future" be encountered by civilians working on jet airliners, the authors recommended some safety measures for persons working on jet fuel cells.

They said the work should be performed by at least two men, with one man outside the cell serving as the observer of the man within the cell. They should carry on a continuous conversation, so that the observer may recognize any emergency and take prompt action in rescuing the repairman.

BOOK REVIEWS

Clinical Recognition and Management of Disturbances of Fluid Balance; by John H. Bland, M. D., 2d ed., Philadelphia, W. B. Saunders Company, 1956, Pp. 522, Price, $11.50.

In the preface to this second edition the author states: "The pyramiding mass of information, both factual and conceptual, in the field of water, electrolyte and hydrogen ion metabolism has made a second edition of this book mandatory." However, of 633 references only 29 were from the literature of 1954-55. Much conceptual information from 1952 to 1955 (the years between the two editions) has been omitted. Although many errors of the first edition have been corrected, there are statements which appear contradictory because the author fails to be explicit.

In striving to provide a practical approach to the problems of disturbances in fluid balance for "clinical utilization at the bedside," the author indulges in great detail, at the expense of repetition, in the recognition and management of these disturbances associated with almost every disease state in all age groups. He includes presentation of the fundamental concepts to the approach of water and electrolyte disturbances followed by discussions of "metabolism" in conjestive heart failure, diabetes, aging, surgery, physical and chemical injuries, and in diseases of the liver, pulmonary system, kidney, adrenals, and nervous system. The concluding chapter touches the problems of magnesium and potassium metabolism. The diagrams in the sections of the book on the fundamentals are clear and helpful to the basic understanding of the text but become sparse in the chapters related to specific disease states.

While the book can be a valuable review and reference for the practitioner, some of the discussions may be confusing to all but the experts in the field. For the teacher it affords an outline source for discussions of the problems in fluid balance. All readers should be aware that there is no empiric approach to the management of electrolyte problems and such a book serves only as a guide to the individual management of the patient.

SAM A. THREEFOOT, M. D.

PUBLICATIONS RECEIVED

Appleton-Century-Crofts, Inc., N. Y.: Modern Perinatal Care, by Leslie V. Dill, M. D.

Grune & Stratton, Inc., N. Y.: Progress in Gynecology, Volume III, by Joe V. Meigs, M. D., and Somers H. Sturgis, M. D.

Paul B. Hoeber, Inc., N. Y.: Dermatologic Formulary, by Frances Pascher, M. D., (2nd Edit.).

The C. V. Mosby Co., St. Louis, Mo.: Handbook of Orthopaedic Surgery, by Alfred Rives Shands, Jr., M. D.

Philosophical Library, Inc., N. Y.: A Book of Contemplation, by Dagobert D. Runes; From Sterility to Fertility, by Elliot E. Philipp, M. A.; Psychopathic Personalities, by Harold Palmer, M. D.

W. B. Saunders Co., Phila.: Current Surgical Management, by John H. Mulholland, M. D., Edwin H. Ellison, M. D., and Stanley R. Friesen, M. D.; One Surgeon's Practice, by Frederick Christopher, M. D.

Charles C Thomas, Publisher, Springfield, Ill.: New Research Techniques of Neuroanatomy, edited by William F. Windle, Ph.D.; Hormonal Regulation of Energy Metabolism, edited by Laurance W. Kinsell, M. D.; Human Blood Coagulation and Its Disorders, by Rosemary Biggs, M. D., and R. G. MacFarlane, M. D., (2nd edit.); Pneumoencephalography, by E. Graeme Robertson, M. D.; The Clinical Aspects of Arteriosclerosis, by Seymour H. Rinzler, M. D.; Mechanisms of Hypertension, With a Consideration of Atherosclerosis, by Henry Alfred Schroeder, M. D.; Hypophysectomy, edited by O. H. Pearson, M. D.; Occipitoposterior Positions, by Edward L. King, M. D.; The Medical Interview, by Ainslie Meares D.P.M.; The Principles of Therapeutics, by J. Harold Burn, M. D.; The Human Brain: From Primitive to Modern, by A. M. Lassek, M. D.; Lens Materials in the Prevention of Eye Injuries, by Arthur Hail Keeney, M. D.; Medical Radiation Biology, by Friedrich Ellinger, M. D.; De Motu Cordis, translated from the original Latin by Kenneth J. Franklin.